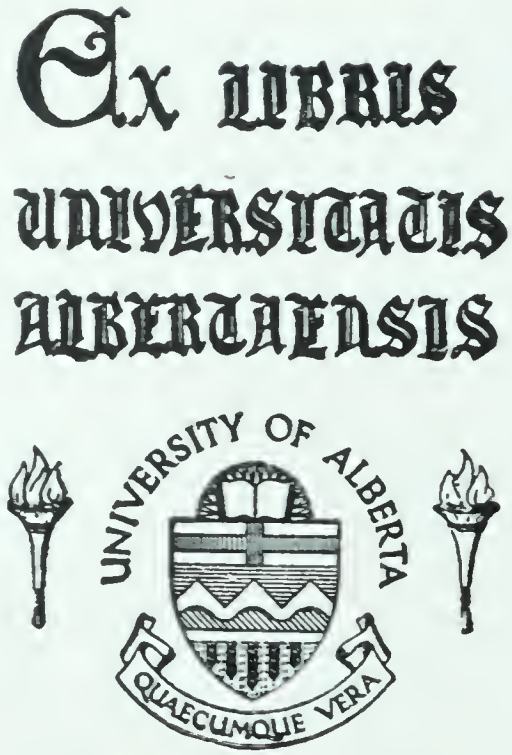
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THE UNIVERSITY OF ALBERTA

AN ELECTROPHYSIOLOGICAL STUDY OF RIGHT ATRIAL MECHANORECEPTORS IN VITRO

BY

JOHN HOWARD PANKHURST

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance a thesis entitled AN ELECTROPHYSIOLOGICAL STUDY OF RIGHT ATRIAL MECHANORECEPTORS IN VITRO submitted by John Howard Pankhurst in partial fulfilment of the requirements for the degree of Master of Science.

ABSTRACT

The Langendorff method of perfusing isolated cat hearts was used in this study of afferent vagal activity from twenty-eight localized right atrial mechanoreceptors.

We were able to show that while the general shape of the adaptation curve was similar to that found by others, the variation was too great to make any specific statements about it. When the amplitude of a step stimulus was increased there was an increase in both peak and adapted frequencies. Similarly, when the velocity of a ramp stimulus was increased the receptor responded sooner, with higher initial frequency and higher peak frequency. The average frequency during the ramp was roughly proportional to the logarithm of ramp velocity.

We were also able to show that the receptors studied exhibited a directional sensitivity to stretch and a model is put forth as an explanation. Tests for linearity were employed and showed that the system was non-linear.

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TABLE OF CONTENTS

	page
ABSTRACT	i
ACKNOWLEDGEMENTS	ii
LIST OF FIGURES	v
INTRODUCTION	1
LITERATURE REVIEW	2
METHODS	10
General	10
Recording Setup	11
Receptor Stimulus	12
Technique of Identification of an Atrial Receptor	12
Local Stretching of the receptor region	14
RESULTS	17
I. Receptor Discharge Under Constant Stimulation	18
Adaptation	
Receptor discharge during different degrees of constant stimulus	19
II. The Effect of Stopped Perfusion Upon Receptor Discharge	21
III. The Effect of Ramp Velocity upon Impulse Frequency	23
IV. Directional Sensitivity to Local Stretching	24
V. Comparison of Dynamic Response with a Static Response	
Theory	26
Results	28
DISCUSSION	30
Adaptation	30
Step Amplitude and Impulse Frequency	31
Stopped Perfusion	31B
Velocity of Stimulus	32
Directional Sensitivity	33
Linearity Test	35
SUMMARY	36

TABLE OF CONTENTS - continued

	page
BIBLIOGRAPHY	38
APPENDIX - Table I	(1)
Table II	(14)
Table III	(28)
Table IV	(41)

LIST OF FIGURES

Figure number	Following page
1	11
2	11
3	17
4 A and B	18
5 A and B	18
6 A and B	19
7	20
8	20
9	22
10 A and B	23
11 A and B	23
12	24
13 A and B	25
14 A and B	27
15 A and B	28
16	30
16'	31A
17	33
18 A and B	34
19	34A

INTRODUCTION

It has been a favourite device of physiologists in studying the function of an organ of the body to isolate it from other bodily influences. Sensory receptors fall into this category and from the first modern attempts and success by Adrian & Zotterman (1926) using frog muscle spindles, *in vitro* preparations have aided the study of sensory receptors.

Recently, stimulus response characteristics of atrial mechanoreceptors have been studied in the frog, cat, dog, and monkey *in situ*. It has been the purpose of this work to develop an *in vitro* preparation using the isolated, perfused cat heart to study right atrial mechanoreceptors, to see if the stimulus response characteristics of these receptors are similar to those obtained by other workers who used *in vivo* preparations, and to obtain additional information about these receptors.

LITERATURE REVIEW

With the advent of the electronic vacuum tube which would respond accurately to potential changes in the magnitude of micro-volts, and magnify the signal until it could be recorded with the available instruments, modern electrophysiology began in earnest. Forbes and Thacher (1920) first used the vacuum tube amplifier to record nerve impulses and with the introduction of the cathode ray oscilloscope by Gasser and Erlanger (1922) physiologists now had a device which was able to faithfully record nerve impulses. Adrian (1926) using a vacuum tube amplifier recorded activity in the vagus nerve of the cat from lung stretch receptors and from cardiac receptors. Realizing the advantages of recording from a single receptor at one time, Adrian and Zotterman (1926) were able to obtain such a preparation in the frog by paring down the sterno-cutaneous muscle until only one active receptor remained. They showed that the greater the velocity of a constant velocity stimulus, i.e., a ramp stimulus, the higher the frequency of discharge of the receptor.

Adrian and Bronk (1928) obtained single receptor recordings not by paring down the muscle containing several receptors as did Adrian and Zotterman (1926), but by paring down the nerve trunk until only one active fibre was left. Matthews (1931), however, was fortunate to find a preparation consisting of the small muscle on the outer side of the middle toe of the frog (m. ext. long. dig. III) in which there was only one mechanoreceptor connected to a single nerve fibre. With it he was able

to study the response of the receptor to mechanical stimuli. He showed that to a maintained step stimulus the receptor would respond with an initial high frequency and would then decrease to a lower value along a definite time course and would remain at this new frequency as long as the stimulus was constant. The receptor would undergo adaptation, as first described by Adrian (1926). Furthermore he also showed that the peak frequency of the receptor was proportional to the logarithm of the stimulus strength. Moreover, if the stimulus was applied with different ramp velocities then the response of the receptor varied in such a way that the greater the velocity the higher the maximum frequency.

A few years later Matthews (1933), also using mammalian muscle spindles and the technique of dissecting nerve fibres developed by Adrian and Bronk (1928), studied the stimulus response characteristics of these receptors. He found that these mammalian receptors were similar to amphibian receptors in a number of ways; for example, different ramp velocities resulted in different rates of rise of impulse frequency, different peak frequencies and different rates of decline of impulse frequency. Specifically, ramps of high velocity yielded rapid increases in frequency to a peak coincident with the end of the ramp, and then a rapid decline in frequency to an adapted frequency. Slow ramps resulted in slow increases in frequency, lower peak frequencies, and slower rates of decline in frequency to the adapted state. However, so long as all the ramps ended at the same final magnitude the adapted frequencies were the same.

Individual cardiac receptors have been studied histologically by Woollard (1926) and Nonidez (1937). Woollard (1926) made a

comprehensive study of the innervation of the heart in which he described sensory receptor endings in the atria of the dog and rabbit. His basis for calling the complex endings he saw sensory was that they innervated a part of the heart that was not muscular and thus could presumably be only of an afferent nature. Eleven years later Nonidez (1937) identified receptor endings in the heart and large veins of the kitten. The maximum zone of concentration of these receptors occurred on the posterior-mesial aspect of the superior vena cava just before it enters the right atrium and in a ring about the inferior vena cava extending into the right atrium, immediately under the endothelium in all cases.

Physiological studies of cardiac receptors have been undertaken by Amann and Shaefer (1943), Whitteridge (1948), Neil and Zotterman (1950), Pearce and Whitteridge (1951), Landgren (1952), Paintal (1953), Struppler and Struppler (1955), Pearce, et al., (1956), Coleridge, et al., (1957), Langrehr (1960), and reviewed repeatedly; the review most recent and most relevant to this work is that by Paintal (1963).

Amann and Shaefer (1943) succeeded in recording activity in multifibre bundles from the cardiac branches of the vagus. They obtained afferent impulses from receptors in the great veins and the atria as well as from lung stretch receptors.

Whitteridge (1948) recorded receptor activity in the cervical vagus from receptors in the heart and lungs. Of particular interest are recordings from receptors thought to be located in the veins leading into the heart which discharge in rhythm with the heart during the 'a' wave of the venous pressure pulse. He also recorded from another receptor having a cardiac rhythm, which he suspected, because of an increase in ac-

tivity during inspiration and a decrease during expiration, to be a pulmonary vascular receptor.

Dickinson (1950), using cardiac branches of the right vagus in the cat, recorded from receptors in the right atrium and was able to show an approximate linear corelation between instantaneous right atrial pressure and instantaneous impulse frequency.

At the same time Neil and Zotterman (1950) recorded afferent activity from both cat and frog vagal fibres of atrial origin. There were two basic types of discharge, one in time with the 'a' wave of the venous pressure curve, which they named type A receptors, and the other in time with the 'v' wave of the venous pressure curve. Pearce and Whitteridge (1951) studied these cardiac receptors with a late systolic burst of activity and concluded that they were likely stretch receptors and that their natural stimulus was distension of the vessels in which they lie.

One of the next advances in the study of cardiovascular mechanoreceptors was with the work of Landgren (1952). Using the isolated perfused carotid sinus preparation from cats he was able to simultaneously record nerve activity from the presso-receptors together with pressure changes in the sinus. From his work a number of valuable conslusions relevant to the present study were drawn. For example, he showed that adaptation in cat carotid sinus receptors followed a power law of the general form $f = fa + k_1 t^{-k}$, where f = impulse frequency, fa = adapted frequency, t = time, k_1 = a constant, and k = a power coefficient.. He also showed that when the pressure inside the sinus was maintained constant, the higher the pressure the higher the adapted frequency. Using a ramp stimulus and varying the velocity of the ramp, Landgren (1952) was able to show that the absolute pressure at which the first spike occurred was lower, the greater the ramp velocity.

Also the greater the ramp velocity, the greater the initial frequency, and the greater the peak frequency.

However, it was not until Paintal (1953) made a detailed study of atrial receptors that specific tests were devised to eliminate receptors showing "adventitious cardiac rhythms" (Whitteridge 1948), such as lung stretch receptors which take on a cardiac rhythm. Furthermore he named the receptors with a late systolic burst, type B. The two main types of receptors were described by him as follows: type A have a short volley of impulses in tyme with the 'a' wave of the venous pressure curve while type B have a late systolic burst of activity in time with the 'v' wave without an 'a' volley. Type B receptors show an increase in activity during an increase in venous return such as that produced by suction on the trachea and a cecrease in activity when positive pressure inflation of the lungs is maintained. Paintal (1953) also localized by punctuate stimulation the region of the receptor endings. These receptors were found on the hind wall in the right atrium, and in heavy concentrations around the openings of the great veins. Paintal (1953) also found that these receptors were relatively insensitive to anoxia. Responses could be obtained from a receptor for about twenty minutes after circulation was stopped. Furthermore he found these type B receptors to be slowly-adapting receptors. When the half time of one of Paintal's (1953) receptors was measured using his data, in Fig. 8, it was approximately 0.1 sec. This is different from Adrian's (1926) classification as his slowly adapting receptors had a half time lof 10 sec., while rapidly adapting receptors had reached zero in 0.5 sec.

Using a vascularly isolated atrium Paintal (1953) injected saline into the atrium via a wide-bore cannula and found that impulse frequency was linearly related to the degree of atrial filling. The impulse frequency was averaged over 0.5 sec. beginning one second after the injection of saline.

Struppler and Struppler (1955) also recorded afferent activity in vagal nerves. They concluded that type A receptors were stimulated by the rate of rise of pressure in the atria.

Coleridge, et al., (1957) combined electrophysiological techniques with those of histology in a study of atrial receptors in the dog. After a receptor had been found and the appropriate recordings made, the heart was opened and the exact position of the receptor determined by punctate stimulation. This area of the atrium was then examined histologically. They found two types of receptors located at the bases of the two cavi and along the hind wall of the right atrium: 1) A syncytial network of fine fibres, 2) thick fibres ending in apparent end structures just under the endocardium about 40 x 50 μ to 250 x 350 μ in diameter. It is this second type of ending which they felt were the receptors responsible for the type A and type B discharge patterns as they were the only discrete structures common to all specimens.

It should be noted that Paintal (1953) had reported a shifting of the discharge pattern of the type B receptors with respect to the e.c.g., during each respiratory cycle.* Later Pearce, et al., (1956) reported that type B receptors could be made to fire a type A discharge upon severe haemorrhage and increased intrathoracic pressure.

Langrehr (1960) recorded from atrial receptors in both cats and

*The shifting of the discharge pattern was attributable to changes in venous return caused by fluctuations in intrathoracic pressure during the respiratory cycle.

dogs. He further separated the discharge patterns into type D and E as well as types A and B. Type D is seen late in the cardiac cycle ending just before the 'p' wave of the e.c.g. Type E is characterized by a short burst between the QRS complex and the T wave of the e.c.g., in time with the salvos from the so-called ventricular receptors. Furthermore Langrehr (1960) has shown that slight changes in the circulatory parameters of the animal can alter the major discharge pattern of the receptor, usually type A or type B, so that most types can be made to fire each salvo type.

When Langrehr (1960) located a receptor, the heart was opened quickly and the receptor located by punctate stimulation. Once located, small forceps were used to take hold of the endocardium on each side of the receptor region, which was then stretched at different velocities by hand while simultaneously recording receptor activity. Langrehr (1960) found that the mean impulse frequency is directly proportional to the logarithm of the stretching velocity and that this relationship held, no matter what receptor type he was recording from.

In a review of vagal afferents Paintal (1963) objects to the idea that there is only one receptor type present in the atria and that it can take on the discharge pattern of any type depending upon the haemodynamic conditions of the animal. Paintal reports that he has been able to record from both types A and B simultaneously in the same chamber and that the type B will show respiratory fluctuations but not the type A.

With the knowledge about right atrial receptors far from complete, it was thought that if an <u>in vitro</u> preparation could be devised,

then, perhaps some of the details unavailable because of the additional complications with in vivo preparations might be obtained from the in vitro preparation. Some of the advantages of the in vitro preparation are discussed later.

METHODS

General

Sixty-one *in vitro* experiments and one *in vivo* experiment were performed using adult cats ranging in weight from 1.8 kg to 4.7 kg. In twenty-four of these experiments, recordings from twenty-eight separate receptors were obtained. In the first twelve experiments the cats were anaesthetised with intraperitoneal injections of nembutal (30 mg/kg), in the next twelve ethyl chloride and ether induction was used followed by intravenous injection of nembutal (30 mg/kg). In each of the remaining thirty-eight experiments, the cat was anaesthetised with ether in a closed box; maintenance of anaesthesia was by nose cone.

At first both the right and left vagi were dissected free in the neck of the cat, together with their common carotid arteries and the sympathetic trunks, which are in the same sheath. Later in the series only the right vagus was removed. A loose ligature was tied around the bundle containing the right vagus, carotid artery, and sympathetic trunk for later identification. The chest of the cat was then opened from the base of the sternum up into the neck with a pair of 8½" Ward dressing scissors. The sides of the chest were spread apart and the heart, lungs, esophagus, trachea, and nerve complex removed, rinsed in 0.9% saline at 37° C, and put in a thermostatically controlled bath of Krebs' solution where the lobes of the lungs were removed and discarded. During the procedure of opening the chest of the cat no attention was

paid to haemostasis since with a little practice the entire operation only took about two minutes from the time of the first incision until the heart was perfused.

The heart was perfused with Krebs' solution at 37° C via the coronary circulation using the Langendorff (1895) preparation. In addition the whole preparation was immersed in a thermostatically controlled bath filled with exchanging Krebs'. The Krebs' solution was aereated with 95% oxygen and 5% carbon dioxide, for the duration of the experiment. The composition of the Krebs' solution used in the experiments was as follows: NaCl-115.48mM/l, KCl-4.63mM/l, $CaCl_2$-4.94mM/l, $MgSO_4$-1.16mM/l, NaH_2PO_4-1.16mM/l. Fresh $NaHCO_3$-21.91mM/l, and glucose 20.00gm/l were added shortly before the experiment began; the pH was about 7.5. The hydrostatic pressure of the perfusate was about 120mm Hg, determined by the height of the perfusion bottle above the preparation. The heart would continue to beat regularly for up to six hours.

Recording Setup

Diagrams of the experimental arrangements used are shown in Figs. 1 and 2. The nerve trunks together with the common carotid artery were lead up onto a platform and pinned loosely to it. With the carotid artery serving as an anchor, the vagus nerve was separated from it for about two centimeters and slipped over a black platform to which the recording electrodes were attached. Paraffin oil was lavered on top of the preparation immersing the recording electrodes and nerve to prevent drying, and to insulate the nerve from ground while recording.

The recording electrodes were attached to the push-pull input of

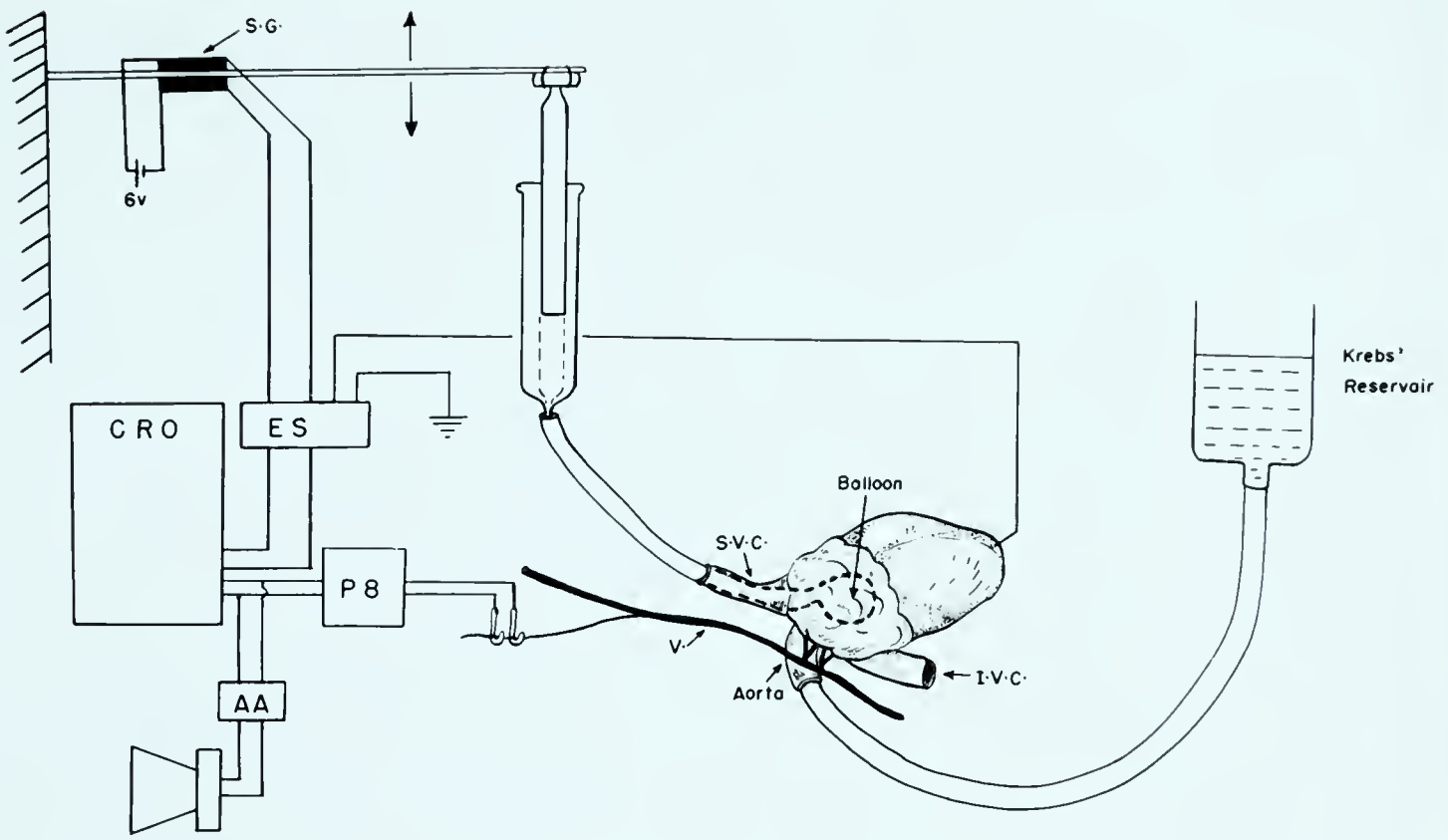


Fig. 1. Diagram of the typical experimental setup using an indwelling balloon as the stimulus. A small bundle teased from the vagus nerve (V) was laid over recording electrodes. Nerve impulses were amplified by the Grass P-8 amplifier (P-8) and displayed on the cathode ray oscilloscope (CRO). Balloon volume was monitored by the strain gauges (S.G.) and their output fed into the electronic switch (E.S.). The e.c.g. electrodes were also attached to the electronic switch and its output from both the strain gauges and the e.c.g. electrodes led into the CRO. The nerve activity was fed from the P-8 to an audio amplifier (A.A.) and a loud speaker.

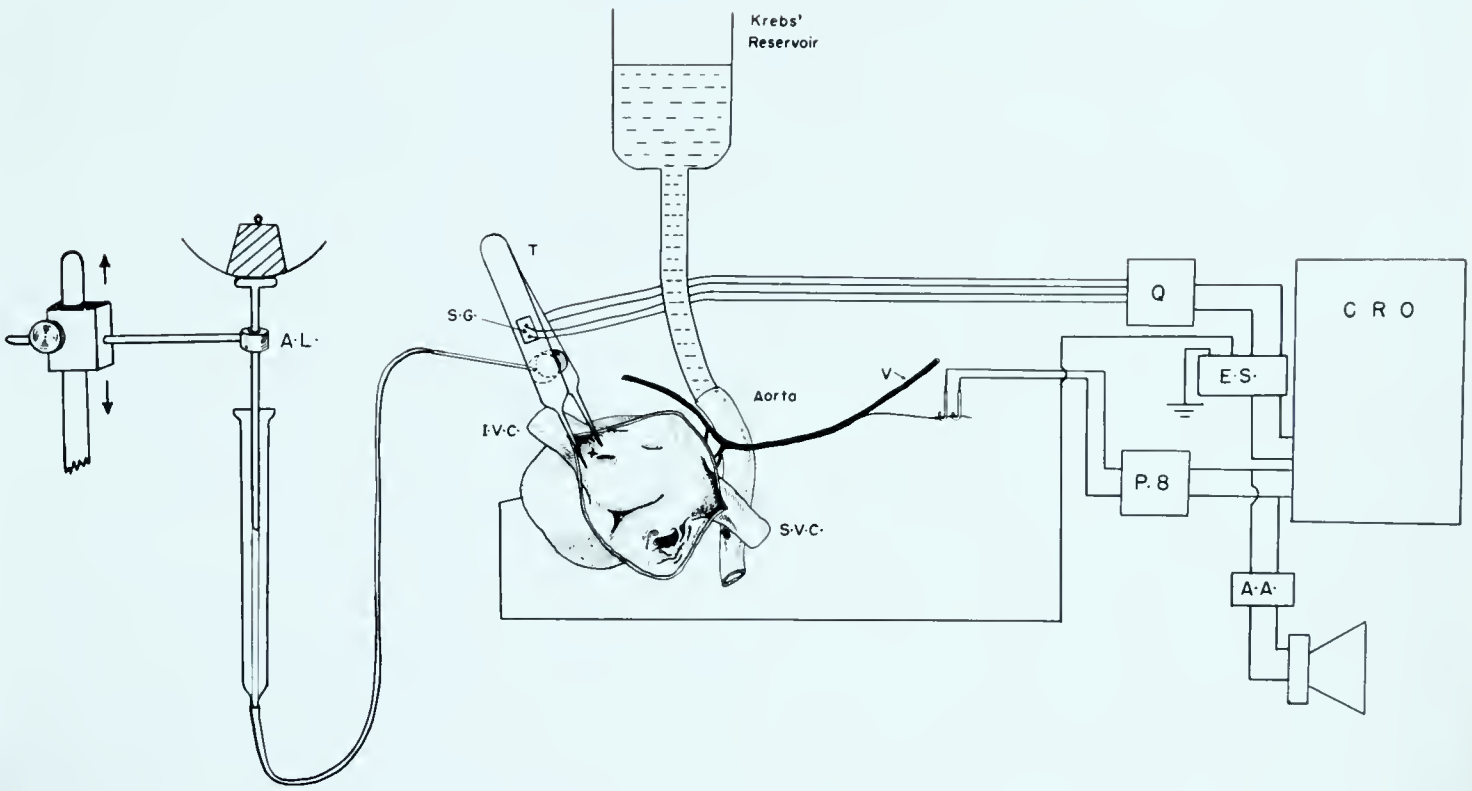


Fig. 2. Diagram of experimental setup for local stretching of the receptor region. The recording system is similar to that in Fig. 1. The right atrium of the perfused heart was slit open and the 'tweakers' (T) mounted over the receptor region (X). A small balloon was placed between the arms of the 'tweakers' and attached to a syringe. A watch glass was attached to the plunger of the syringe so that a weight could be placed on top of the plunger to depress it. The amount of stretch applied to the receptor region was controlled by means of the adjustable limit (A.L.).

an A.C. Grass P8 preamplifier. The output of the preamplifier was fed into the upper channel of a two channel cathode ray oscilloscope (Tektronix 502). The bottom channel was split using a conventional electronic switch, and used to monitor the e.c.g. and the stimulus. The e.c.g. was measured by simply piercing the apex of the ventricle with one end of a fine wire, insulated except for the tip, and measuring changes in potential between this point and ground.

Receptor Stimulus

Balloons made of condom rubber, attached to the end of a flared polyethylene catheter, were inserted into the right atrium either through a slit in the atrial appendage or through the cut end of the inferior vena cava (Fig. 1). The other end of the catheter was attached to a 10 ml syringe so that the balloon could be inflated with a measured amount of air. After the first few experiments it was found useful to know the rate at which the balloon was inflated. To measure this, the plunger of the syringe was attached to one end of a stiff steel strap. Near the anchored end four Baldwin-Lima-Hamilton, Sr-4, Type A-1 strain gauges were attached; these were connected as a Wheatstone bridge. When the plunger of the syringe was moved, the change in potential across the bridge formed by the strain gauges, which was proportional to the displacement of the plunger, could be monitored on the cathode ray oscilloscope. Receptor discharge was recorded together with different rates of application of the stimulus and different absolute amounts of stimulus.

Technique of Identification of an Atrial Receptor

The technique of isolating single functional units from the right

cervical vagus was essentially that described by Whitteridge (1948). The technique of dissecting 'single' fibres from the vagal trunk is similar in both the *in vivo* and *in vitro* experiments as well as the mode of stimulation of the receptors using an indwelling balloon.

The method of finding right atrial mechano-receptors was as follows: a small bundle of nerve fibres was placed on the recording electrodes and the right atrial balloon inflated to 10 ml. If there was no discharge in the bundle a new one was tried until receptor activity could be detected upon inflation of the balloon. Once a receptor discharge could be seen the receptor was localized by probing with a fine glass rod. It was important to do this before recording any data as there are tracheal, bronchial, and esophageal receptors present in the preparation which can be stimulated when the balloon is inflated, and with the heart beating they can all take on a cardiac rhythm. It is also possible that the receptor may be located in any of the chambers of the heart other than the right atrium. For example, one receptor was stimulated when the balloon was inflated and from the appearance of the discharge pattern it was assumed that it was located in the right atrium. After a series of records were obtained the receptor was finally localized by punctate stimulation in the right ventricle. What had happened was, the balloon when inflated had bulged into the right ventricle through the tricuspid valve and had stimulated the receptor.

There are some advantages in using an *in vitro* preparation such as this and it would be worthwhile mentioning some of them here; however, it should be remembered that the results obtained in this way are not necessarily the same as in the whole-animal experiments. In the *in vitro*

experiments there are no problems with bleeding to obscure the dissecting field, and any leaks in the system can easily be tied off so that perfusion of the coronary vessels can be maintained. The necessity of dissecting the vagus until only one functional unit remains is often unnecessary as many of the afferent fibres in the trunk have had their connections with their receptors severed, in particular pulmonary and bronchial fibres, so that not as many multi-unit bundles are encountered as in the whole animal experiments. If there does happen to be more than one receptor active in the same bundle of nerve, say for example a right atrial receptor and a tracheal receptor, both of which fire during the inflation of the balloon, all that is necessary to make it a single unit of atrial origin is to locate the tracheal receptor by punctate stimulation and pinch the receptor region with fine forceps destroying it. In the whole animal the nerve bundle would have to be removed from the electrodes and the two active fibres separated by further splitting of the bundle which, of course, endangers losing both of the receptors.

Paintal (1953) found that these receptors were relatively insensitive to anoxia and that they would respond for about twenty minutes after the animal was killed. This meant that any manoeuvers to be done on the localized receptor had to be done within this time, whereas in this preparation you have on the average six hours.

Local Stretching of the Receptor Region

First receptor was located by punctate stimulation with a small diameter glass probe. A glass rod was then inserted into the atrium via the inferior vena cava, through it and out the superior vena cava. A

pair of scissors was used to cut open the atrium along the top of the glass rod. The atrium could now be laid open and held firmly so that the exact location of the receptor could be made and noted.

The device used to stretch the receptor region consisted of a pair of jeweller's hair-spring forceps, hereafter called 'tweakers'. Each tip was modified to act like a pair of small tissue clamps (Fig. 2). Four Baldwin-Lima-Hamilton, SR-4, Type FAP-25-12 foil strain gauges were mounted, one on each side of the two arms of the forceps, and operated similarly to those on the steel strap. The forceps were mounted on a micrometer arm of a "stereotaxic" apparatus so that they could be moved horizontally, vertically, and rotated 360° about the vertical axis. A small, flat balloon about 2 - 3 mm thick was put between the arms of the forceps and attached to a tuberculin syringe by a catheter. The system was filled with water. To spread the tips of the forceps all that was necessary was to depress the plunger of the syringe. To do this at constant rates a platform was attached to the top of the syringe so that weights could be dropped onto it (Fig. 2). Ideally, in such a system, when the weight is placed on top of the syringe and released, the displacement of the plunger, and therefore the separation of the tips of the forceps, exhibit an exponential time course. Since what is required is a constant-velocity time course, the weights chosen to depress the plunger were great enough to make the first part of the exponential decay (nearly linear part) long enough to open the 'tweakers' the required distance. To adjust the degree of elongation an adjustable limit was also important to keep the operation in the first part of the exponential course before it gets non-linear. Different ramp velocities were obtained by increasing

the flow resistance of the catheter between the balloon and the syringe with a small clamp. The beginnings of the ramps were difficult to generate properly due mainly to friction between the plunger and the barrel of the syringe (see Fig. 7). To help overcome this, immediately before each run a drop of water was applied to the top junction of the plunger and the barrel of the syringe, and then the plunger was spun by hand, to minimize starting friction.

The strain gauges were attached to a strain gauge amplifier (Type Q Tektronix) whose output was displayed on the cathode ray oscilloscope. Recordings were made of the nerve discharge at different degrees of stretch of the receptor region, at different rates of deformation, and in different directions of stretch. The output of the strain gauges in both the balloon and 'tweaker' experiments was linear. Permanent records were simultaneously made of the e.c.g., the time course of the stimulus, and of receptor discharge, by photographing the oscilloscope display with moving recording paper in a Grass C-4 Kymograph Camera at a paper speed of 100 mm/sec.

RESULTS

Of the sixty-one perfused heart preparations studied, nineteen produced useful results. In these nineteen successful experiments twenty-five right atrial receptors were localized by punctate stimulation (Fig. 3). Stimulation of eighteen receptors was carried out using an indwelling balloon, and of eight by local stretching with the 'tweakers' described above. In one case successful stimulation by both methods was performed. In the first four experiments using an indwelling balloon no record was made of the stimulus; however, in all of the remaining experiments the stimulus was recorded.

In analysing the results two main criteria were kept in mind: first, to compare our results with the work of others who had performed experiments on related mechanoreceptors, and second, to try to obtain some additional information about these cardiac receptors. To facilitate this the data have been discussed under different sections as follows: I. Receptor discharge during constant stimulation, under which adaptation, and the effects of stimulus strength were studied; II. The effect of stopped perfusion upon receptor discharge; III. The effect of ramp velocity upon impulse frequency; IV. Directional sensitivity to local stretching; V. The response during ramp stimulation was compared with the time integral of the response during the period of constant stimulus following the ramp. Some of the theory is presented followed by the results.

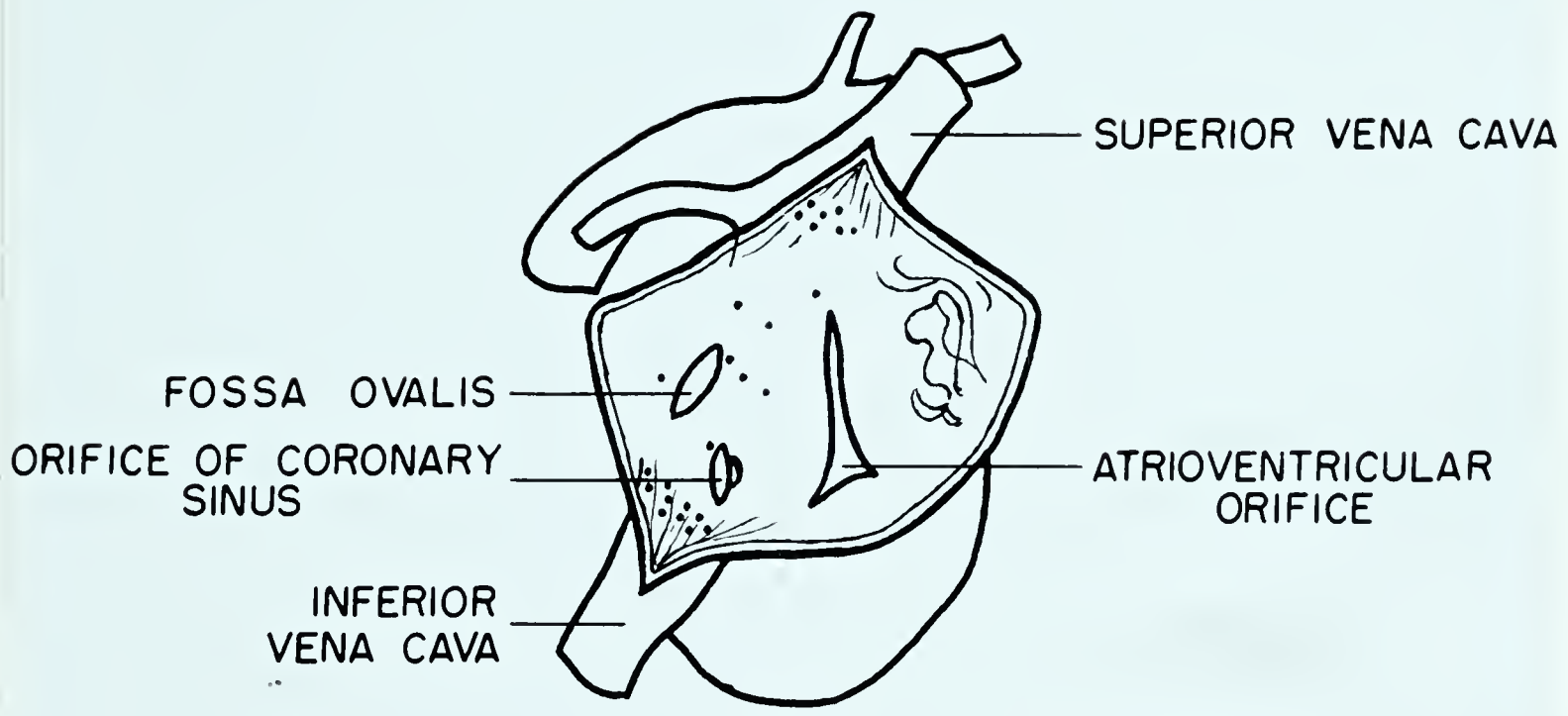


Fig. 3. Schematic diagram of a heart with the right atrium opened (after Field and Taylor, 1950). The dots represent the approximate positions of 25 receptors localized by punctate stimulation in 19 hearts.

I. Receptor Discharge Under Constant Stimulation

Adaptation

When a right atrial receptor was stimulated by rapid inflation of an indwelling balloon, or by rapid stretching of the receptor region to a degree great enough to result in a maintained discharge of the receptor, there results a characteristic burst of activity from the receptor (Fig. 4, A and B respectively). If the stimulus is applied rapidly enough, the impulse frequency rises quickly to a maximum and then when the stimulus is maintained constant it falls along a definite curve of the general shape seen in Fig. 5 A and B. One useful way of describing how fast a receptor adapts is by a measure of its half time, the time it takes for the frequency to drop to one-half of its peak frequency. The half times for the two receptors shown in Fig. 5 A and B are 0.25 sec. and 0.025 sec. respectively. The half times for the other receptors studied have been calculated and appear in Table 1. It should be noted that the half times recorded for each receptor vary with each run. This is really not surprising since the response of a receptor is intimately related to its past history, which, in this case, would be the length of time since the last stimulus, and most important, the rate with which the stimulus was applied and its final absolute magnitude.

With both modes of stimulation, transient changes in impulse frequency accompany each normal contraction of the myocardium. Examples of this cardiac modulation in output of the receptor can be seen in Figs. 4 and 5.

The responses shown in Fig. 4 A and B were replotted on semi-log and log-log paper to see if the decline in impulse frequency followed

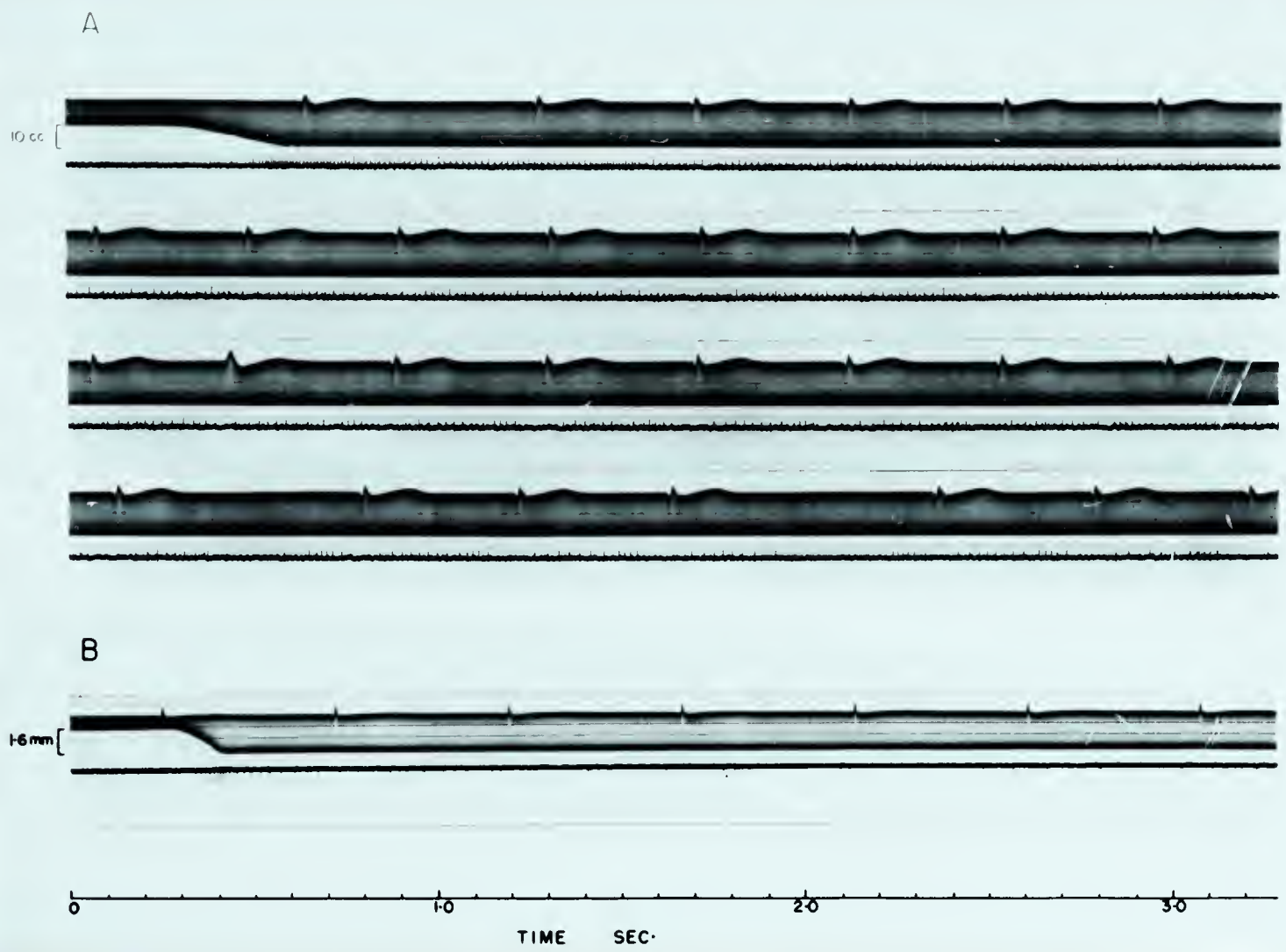


Fig. 4. A) Continuous recording from an atrial mechanoreceptor when being stimulated by inflation of an indwelling balloon. Reading from top to bottom on each filmstrip the traces record: e.c.g. (upper edge of gray region), balloon volume (lower edge of gray region), and nerve impulses. The gray region represents the electronix switch artifact.

B) Recording from another atrial mechanoreceptor stimulated by local stretching of the receptor region.



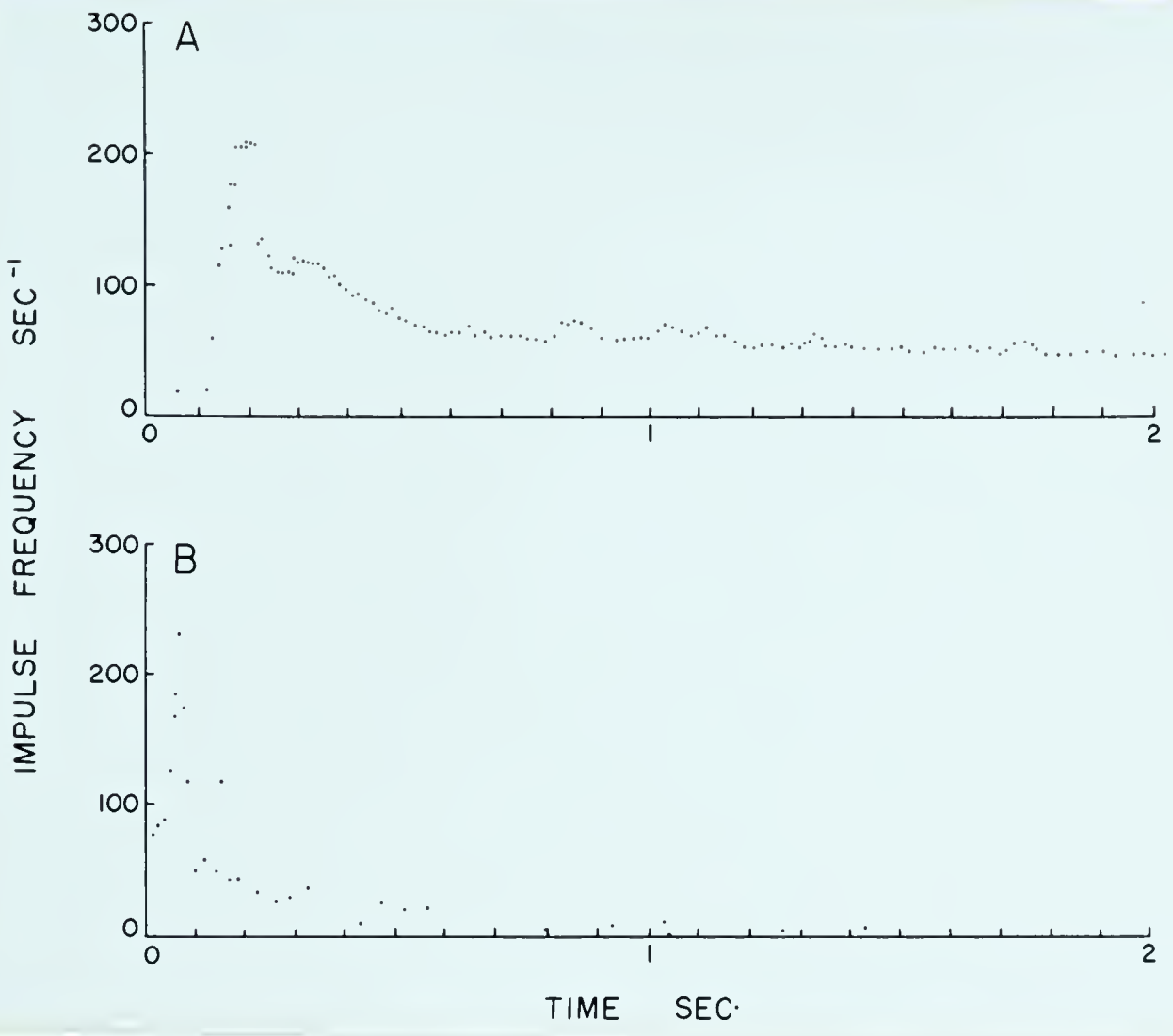


Fig. 5. Impulse frequency is plotted on the ordinate as the reciprocal of the time interval between successive impulses against time on the abscissa. Time zero was taken as the time of the first impulse in each case. Cardiac modulation is easily seen as slight bumps in these curves. Curve A is drawn from the first strip of the record in Fig. 4 (A) and curve B from the record in Fig. 4 (B). They show the time course of the rapid rise and subsequent decay in frequency of the receptors.

an exponential or a power decay respectively. The results of this plot are shown in Fig. 6 A and B. Estimating by eye it seems that the decay in frequency with time follows a power law. However, the records are not compelling and while both Landgren (1952) working with carotid sinus baroreceptors and Chapman and Smith (1963) working with cockroach mechanoreceptors found a power law relationship one cannot be certain of such a relationship with the data available from the work reported here. In spite of this we attempted to determine the slope of the log-log plot where it was possible (Table 1). The general equation of a power function is $f = at^{-k}$, where f is the frequency, t is the time, k is the slope, and a is a constant. The values of k that were calculated from the graphs vary with each run when the stimulus parameters are changed. The values of k calculated from the curves in Fig. 6 A and B are 0.48 and 0.02 respectively.

Receptor discharge during different degrees of constant stimulus

In order to study the effects of stimulus amplitude upon the response of the receptors the magnitude of the applied stimulus was varied in the following way: In the experiments using an indwelling balloon to stimulate the receptors, the balloon was inflated in 1 ml increments up to 10 ml. Each run would start with the balloon collapsed and then inflated to a predetermined volume at constant velocity and held constant for approximately 10 sec. during which the discharge of the receptor could be recorded. Similarly in the localized stretching of the receptor region, the length of the myocardium containing the receptor was varied. Slack length between the tips of the stretching forceps

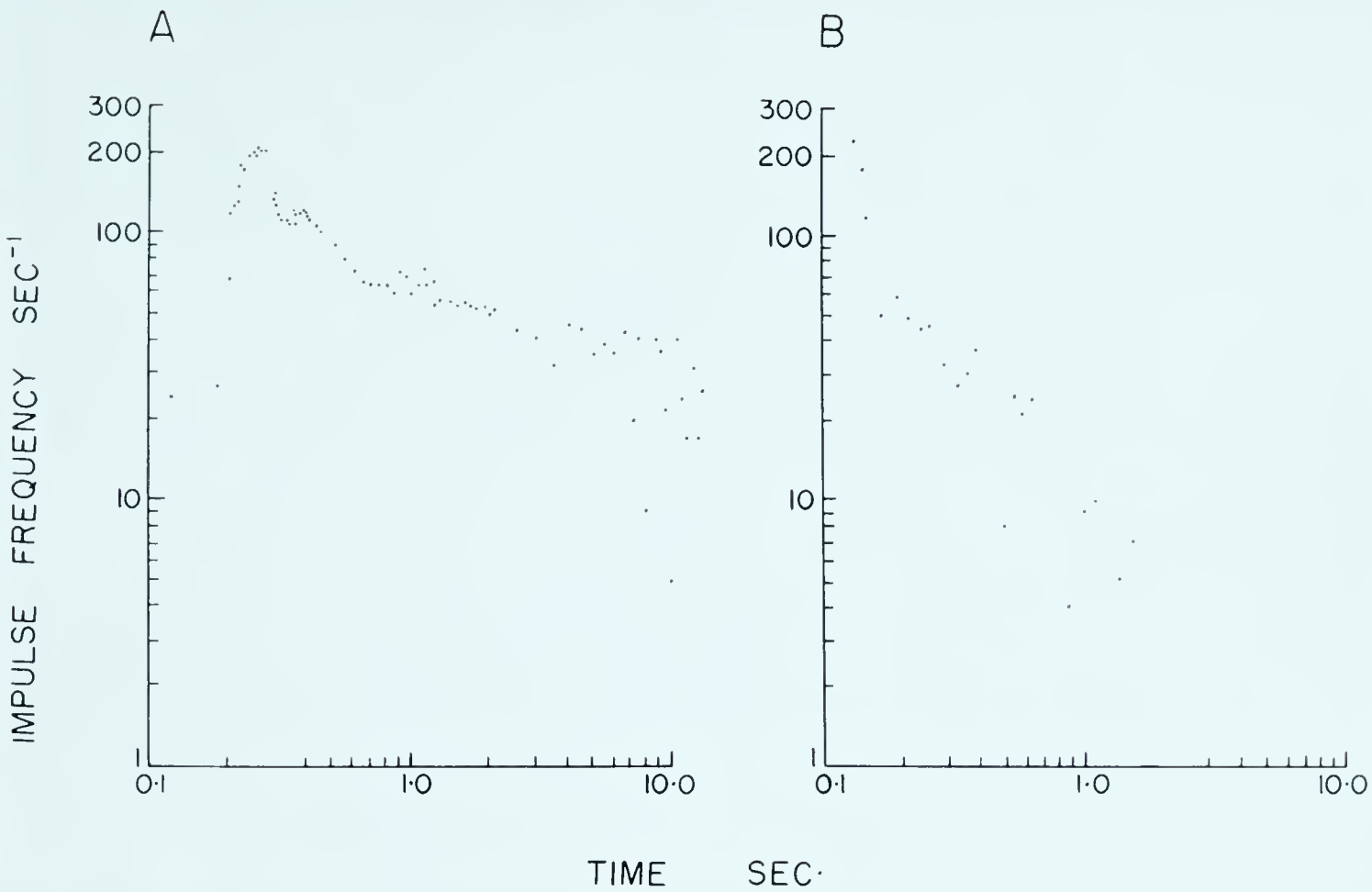


Fig. 6. A and B. Log-log plot of the data from Fig. 4 A and B. Time zero was taken as in Fig. 5.

corresponds to zero stretch, or rest length. The increase in length between the tips of the forceps was recorded with the receptor discharge. The percentage increase above rest length was calculated as percent elongation. Part of a series of such an experiment is shown in Fig. 7 and in the graphs of impulse frequency against time it is possible to see the effect of stimulus amplitude on frequency of discharge of the receptor (Fig. 8).

We were unable to show conclusively that the peak frequency of a receptor during each run increases with the magnitude of the stimulus. In a series of experiments in which the stimulus was applied quickly so that it approximated a step stimulus, some results(but not all) show that as the amplitude of the step increases, so does the peak frequency. One reason for this disparity is that we were unable to duplicate the same ramp precisely from one trial to the next. While the absolute amount of elongation applied to a receptor region could be controlled, the static and dynamic friction between the plunger and the barrel of the syringe made it difficult to control the velocity with which the stimulus is applied and as will be seen in section III, the ramp velocity affects the peak frequency.

Matthews (1931), Boyd and Roberts (1953), and Loewenstein (1956) all working with mechanoreceptors, have shown that the adapted frequency of a receptor is related to the amplitude of the stimulus in such a way that the higher the stimulus the greater the adapted impulse frequency. In the majority of the experiments performed the completely adapted frequency was not available, as in some cases the records were too short, or in the majority of cases, the records became pulsatile and meaningful

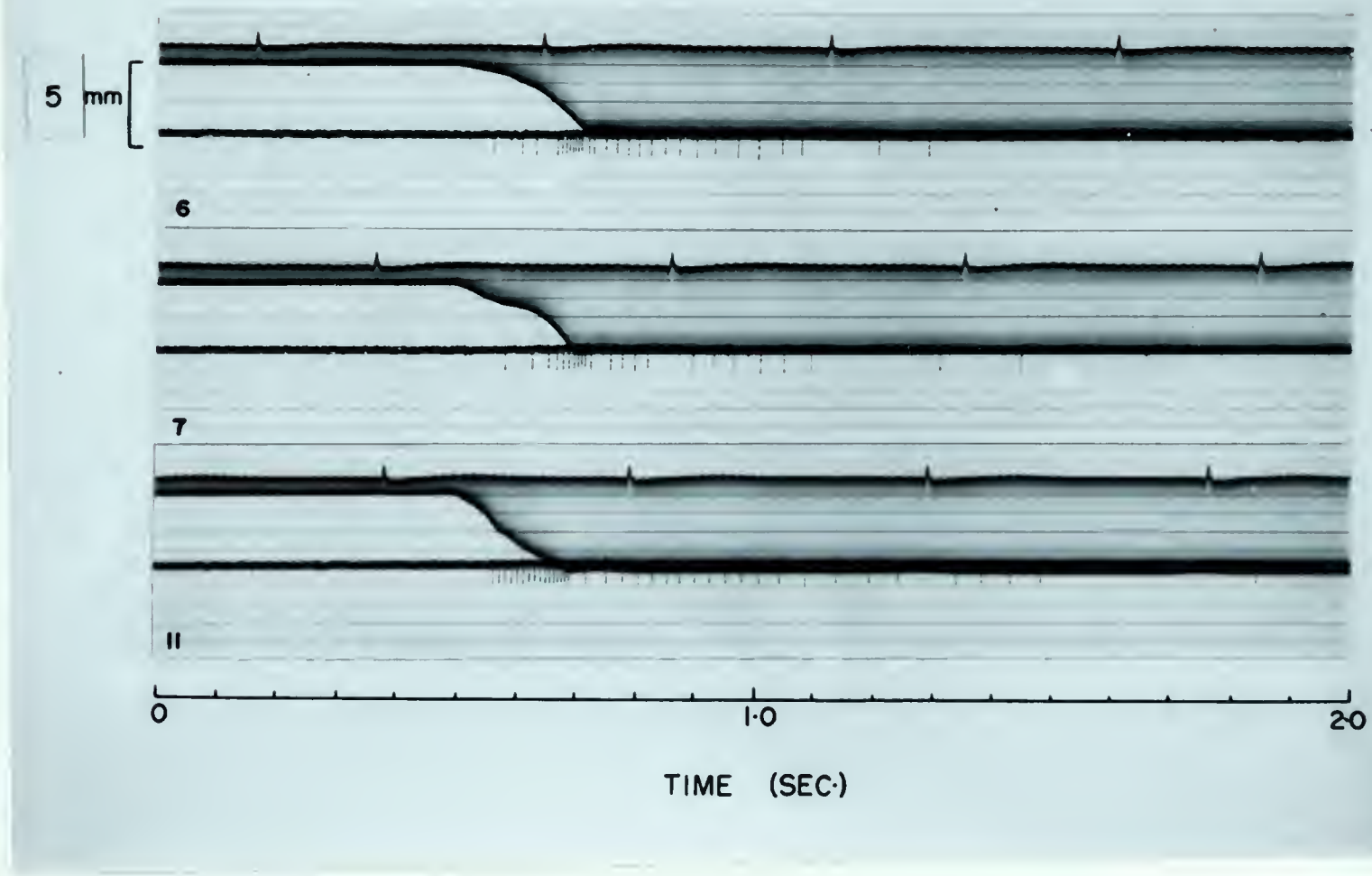


Fig. 7. Film record of three rapid local stretches, Run 6, 7 and 11 of experiment 59, receptor 63. The final magnitude of stretch was slightly different for each run. The records on each filmstrip are as in Fig. 4.

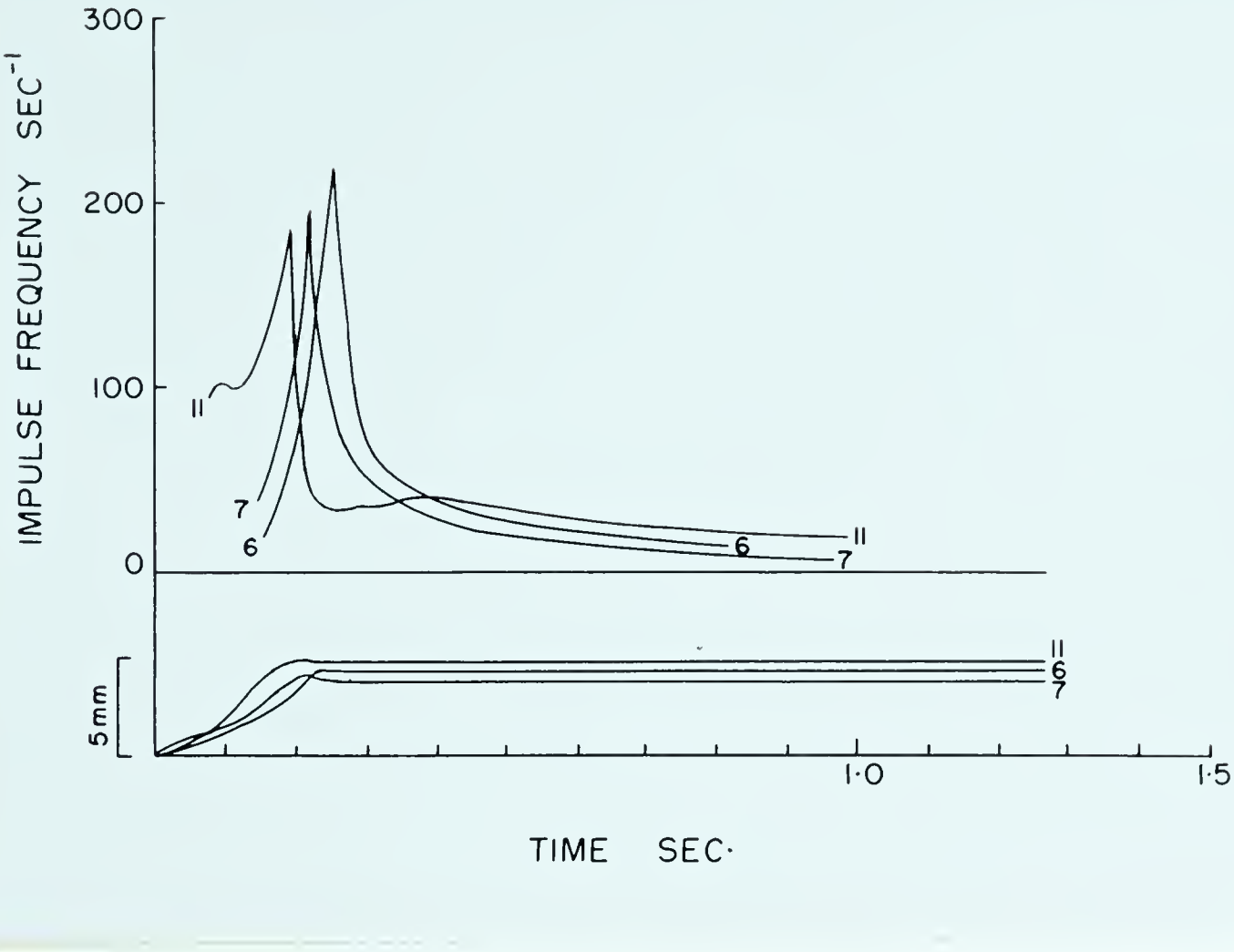


Fig. 8. Graphs of impulse frequency vs time for the data of Fig. 7. The time course and magnitude of the three separate stimuli are shown at the bottom of the graph.

interpretation of them was not possible. Because of this the partially adapted frequency of the receptor was taken. The time used for this frequency was either the frequency one second after the stimulus became constant or one second after the start of the stimulus. Because the half times of the receptors are generally less than 0.30 sec. in most cases the discharge frequency is no longer changing rapidly at the one second mark so that a valid comparison can be made. The results in Fig. 8 are typical of the majority of receptors and are consistent with the work of others, that is, the higher the stimulus amplitude, the greater the adapted or partially adapted frequency.

II. The Effect of Stopped Perfusion Upon Receptor Discharge

In the course of his experiments in which he located the exact position of atrial mechanoreceptors by punctate stimulation, Paintal (1953) found that these receptors were relatively insensitive tc anoxia. He found that a response could be elicited from these receptors for some twenty minutes after arrest of circulation *in vivo*. However, Paintal did not report whether during this twenty minutes there was a slow decline in activity or whether the response of the receptors studied remained similar to that before the circulation was clamped until near the twenty minute period, with a final rapid deterioration in the preparation. The purpose of these experiments was to see if the *in vitro* preparation would act the same way and also to try and get some further information on how the receptor discharge changed with time after perfusion was stopped.

In one experiment, three hours and twenty-five minutes after the heart had been removed from the cat and was being perfused in the usual

manner, stopped perfusion studies were done. Two control runs were made using an indwelling balloon to stimulate the receptor and than at time zero the perfusion line was clamped. At varied intervals after this the response of the receptor was sampled. The peak response of the receptor, and the frequency five seconds after the start of the inflation, were plotted against time after the perfusion had been stopped (Fig. 9). It can be seen that the receptor responded for twenty minutes, during which there was little variation in both peak frequency, and partially adapted frequency. The next application of the stimulus at twenty-five minutes did not result in a response. The perfusion line to the heart was opened at twenty-six minutes, and within nine minutes the receptor was once again capable of responding to a stimulus.

During the stopped perfusion manoeuvre it was also noted that the heart stopped beating spontaneously fourteen minutes after the perfusion was stopped. However, while there was no obvious contraction of the myocardium with the naked eye, the response of the receptor remained pulsatile. Six minutes after perfusion was started again the heart started to beat spontaneously. During the course of another experiment on a right ventricular receptor, there was little change in the discharge pattern upon stopped perfusion for thirty minutes. After this there was a slow deterioration in the response until forty-five minutes after the perfusion was stopped the receptor would only fire one or two impulses upon each inflation of the balloon. After fifty-three minutes the receptor did not respond to stimulation. Opening the perfusion line resulted in a partial recovery of the receptor. In preliminary experiments when the isolated heart was not perfused, the vagus nerve remained excitable to electrical stimulation for at least three hours. Thus it is not likely to be the nerve which was responsible for cessation of activity upon stopped perfusion.

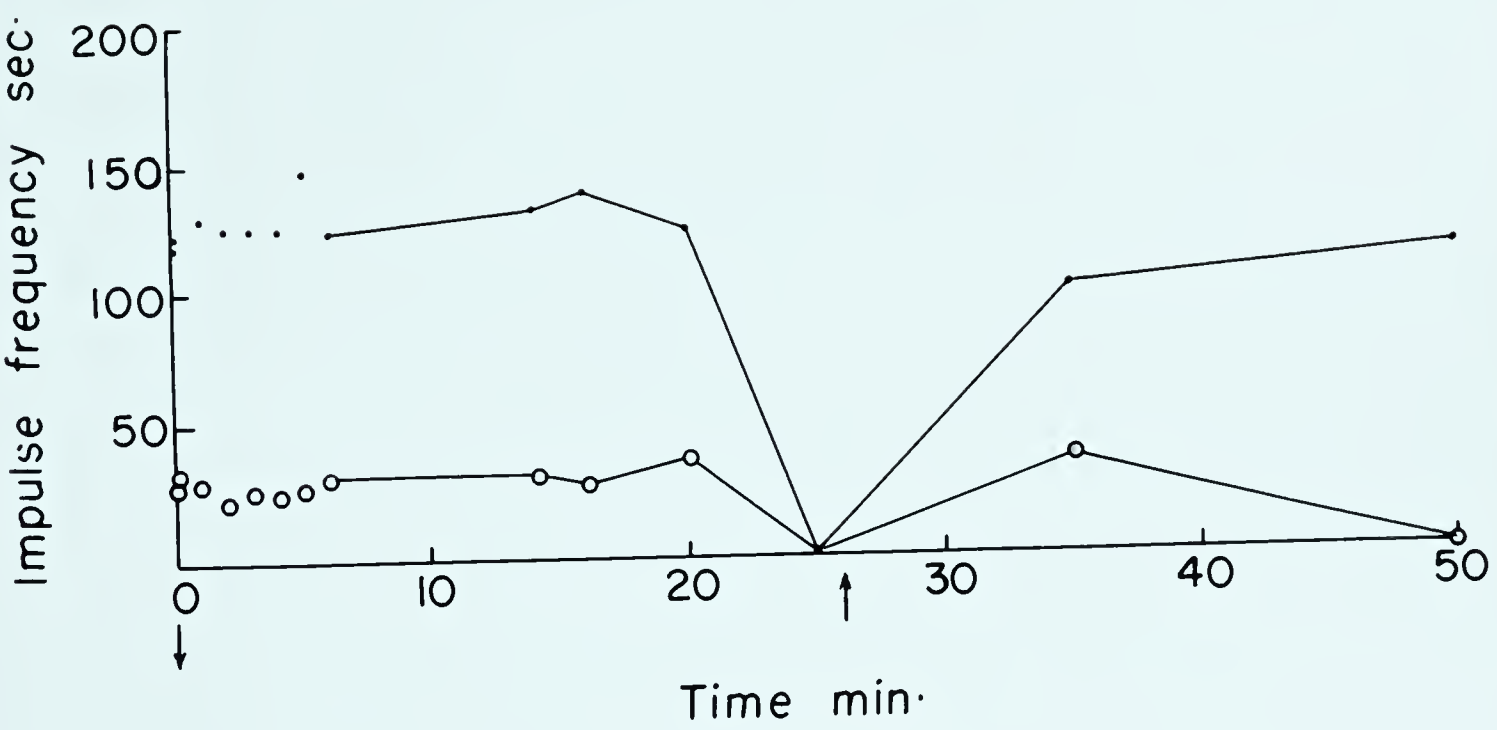


Fig. 9. Peak impulse frequency (.....) and the impulse frequency 5 sec. after the beginning of successive 8 ml balloon inflations, each 10 sec. in duration (oooo), are plotted against time during and after a period of stopped perfusion for experiment 18, receptor 3. The perfusion was stopped at the downward arrow and started again 26 min. later at the upward arrow. Previous control levels for two tests are shown at time zero.

III. The Effect of Ramp Velocity Upon Impulse Frequency

It is to be expected that the response of a receptor which adapts will vary with the rate at which a stimulus is increased. In these experiments an attempt was made to apply a stimulus which increased linearly in time. However, this proved very difficult to do by hand, and at best the start and end of these approximate ramp stimuli were rounded.

Fig. 10 A and B shows the response of two right atrial receptors to ramps applied by increasing balloon volume linearly and by local stretching at a constant velocity.

It should be remembered that when the stimulus is applied by inflating a balloon there is the problem of how the balloon is coupled to the atrial wall. The geometry of the balloon and the atrium also contribute to this problem. For example, when large balloon volumes are reached the balloon will form an extension out into the right ventricle through the tricuspid valve or up or down what remains of the two venae cavae. Thus while the balloon is fully inflated the receptor regions are not stretched to the extent which one would expect. For this reason it is unlikely that a 10 ml. balloon inflation would have the same effect on two different receptors in the right atrium particularly when these are in different regions of the atrial wall. This limits the comparisons that can be made between the impulse frequency of different receptors.

To see more clearly how different velocities of inflation alter the impulse frequency the data in Fig. 10 was plotted showing impulse frequency vs. time in Fig. 11 A and B. In both series of experiments the following information is evident. The greater the rate of rise of the ramp the sooner the first impulse occurs and the greater the peak

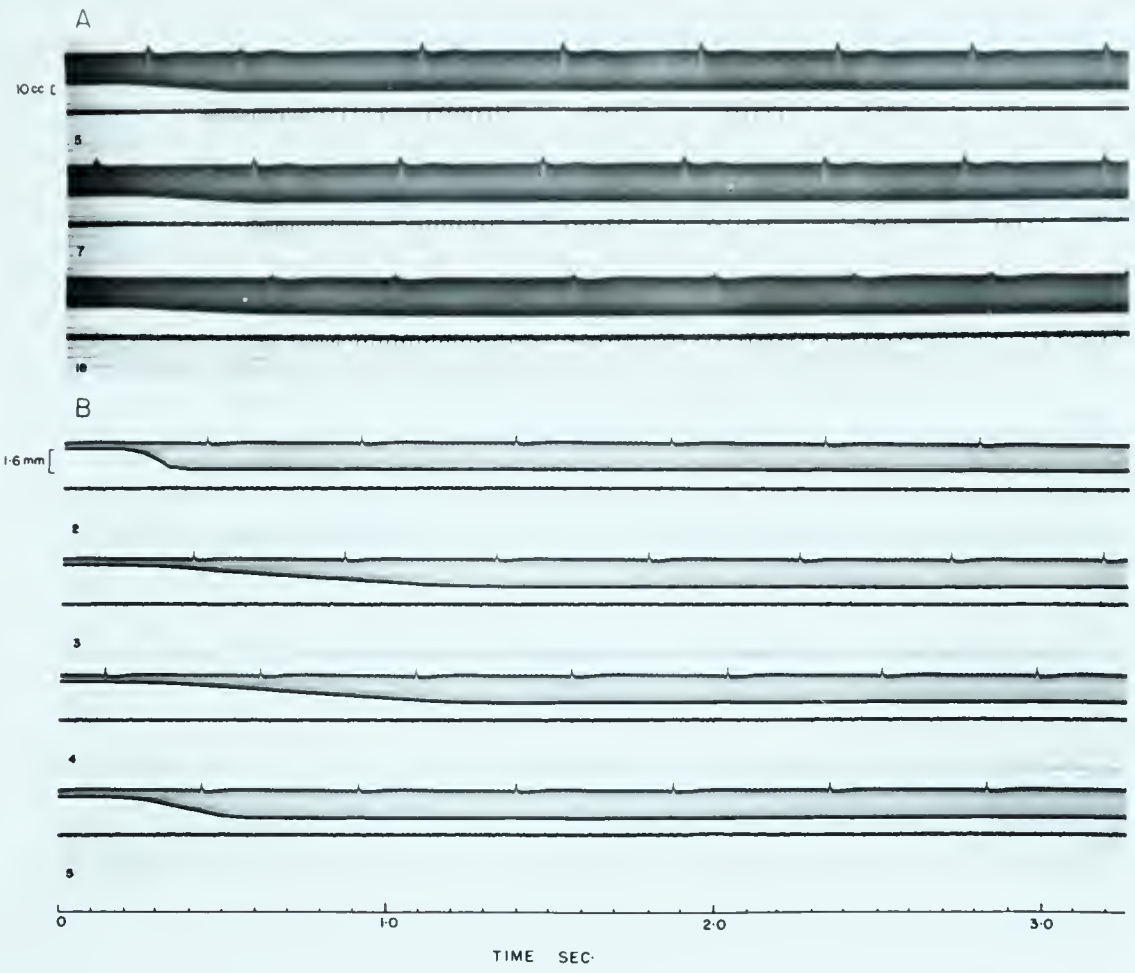


Fig. 10. A) Indwelling balloon experiment 52, runs 5, 7 and 18. Ramp velocity was varied but the final balloon volume was constant.

B) Local stretch experiment 59, runs A2, 3, 4, 5. Again as in A above, ramp velocities were varied but the percentage elongation of the receptor region was kept constant.

Traces as in Fig. 4.

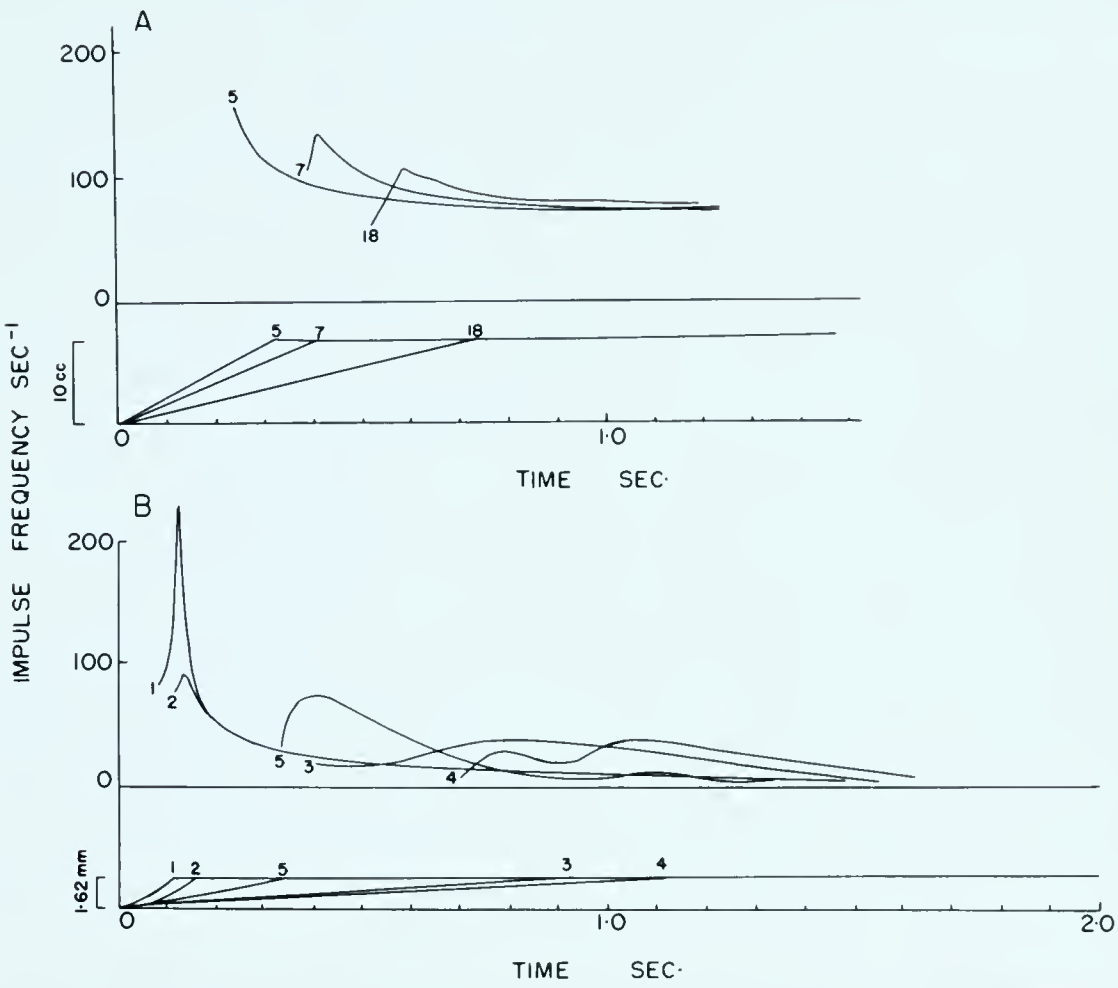


Fig. 11. A) Impulse frequency as a function of time has been plotted for three runs, 5, 7 and 18, all from experiment 52, receptor 57. Beneath this is a plot of ramp velocity.

B) Data from 'tweaker' experiment 59, receptor 63, runs 1, 2, 3, 4, and 5 have been plotted as in A).

frequency. There are exceptions to this, but in the majority of cases this is true (Table III). While this is true during the ramp, the impulse frequency one second after the stimulus becomes constant, seems relatively insensitive to the ramp velocity, and is mainly dependent upon the absolute magnitude of the stimulus. This can be seen by comparing the results shown in Fig. 11 A and B, where the different ramps all end at the same value, with those in Fig. 8 where they end at different magnitudes.

IV. Directional Sensitivity to Local Stretching

Nonidez (1937) and later Coleridge, et al. (1957) described these receptors from histological studies as being within the endocardium or in the connective tissue space between the endocardium and the myocardium of the right atrium. As mentioned earlier they found that the nerve fibres ended in enlargements which were about 40 x 50μ to 250 x 350μ in diameter and arranged parallel to these muscle layers. In one typical experiment we removed the region of the right atrium where a receptor had been localized by punctate stimulation. The tissue was fixed, and sectioned and measurements made from the resulting slides were: endocardium 18-25μ; connective tissue 10-12.5μ; myocardium 333-540μ (Fig. 12). From these measurements it is very likely that the 'tweakers' were attached deeply enough into the tissue to elongate the actual receptor region by the amount measured.

Because the receptor region with which we are dealing is near the inner surface of the atrium a partially inflated balloon was used as a model atrium, to gain some insight into what the 'tweakers' might be doing to the atrial wall. A grid was stamped on the balloon and this

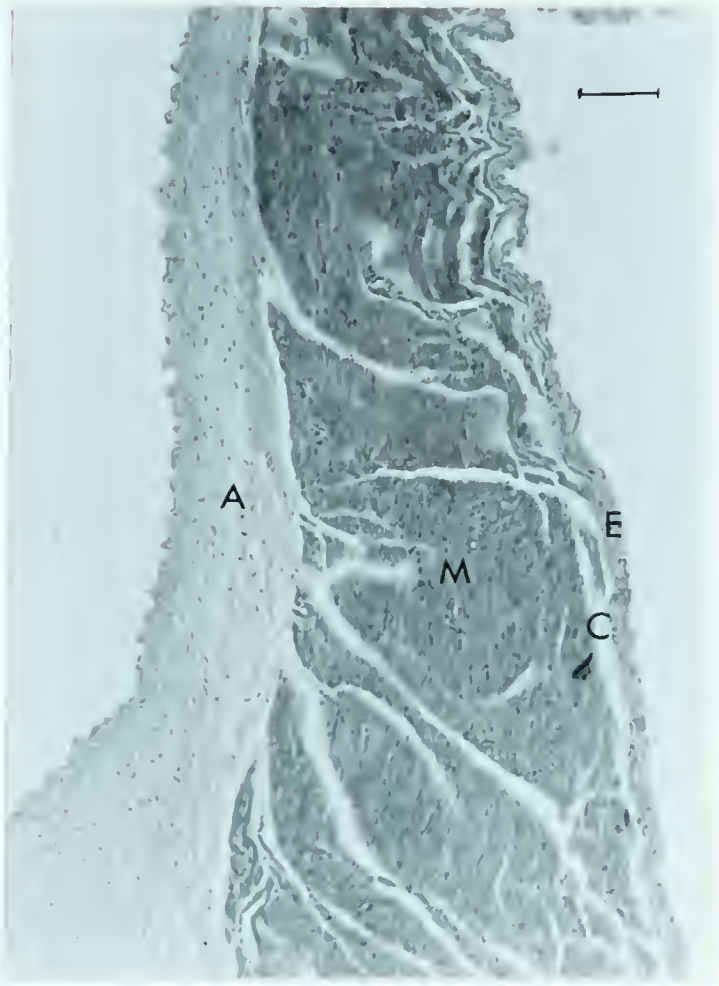


Fig. 12. Haematoxylin and eosin stained transverse section through a 2 mm square piece of atrial wall in which a receptor had been localized and studied, to show the relative thickness of components of the atrial wall. No stain for nerve endings was used.
A - epicardium
M - myocardium
C - connective tissue
E - endocardium
Calibration 100μ.

region deformed by hand with tweezers. When the balloon was stretched between two points, which were a comparable distance apart as the two tips of the 'tweakers', the grid became compressed perpendicularly to the direction of elongation. While these deformations of an elastic sheet are only a crude model of the atrial preparation, they point out the probable nature of the 'tweaker' stimulus and suggest a probable basis for directional sensitivity.

Keeping in mind the difficulties of knowing how a receptor is actually deformed, a series of experiments was performed to study directional sensitivity in which two or more directions were used. The experiments done are listed in Table IV together with the directions of stretch.

In one experiment three directions of stretch were used. The results were plotted on a semi-log graph and show initial frequency vs time and peak frequency vs time respectively (Fig. 13 A and B). While no details about the way in which the receptor is arranged are available it is obvious from these graphs that there is definitely a directional sensitivity. For example, in one direction the threshold of the receptor is reached at a stimulus velocity of less than 0.69 mm/sec. while in another direction threshold velocity is about 6.0 mm/sec. Peak frequency also varies depending upon the direction of the deforming stimulus.

The absolute amount of stretch applied to the receptor region also resulted in a varied output depending upon the direction of the deformation. For example, with the stimulus rates of between 15 to 25 mm/sec. at 20.8% increase in length, one direction of stretch yielded a strong volley, while when stretched in a direction 90° to this, a similar

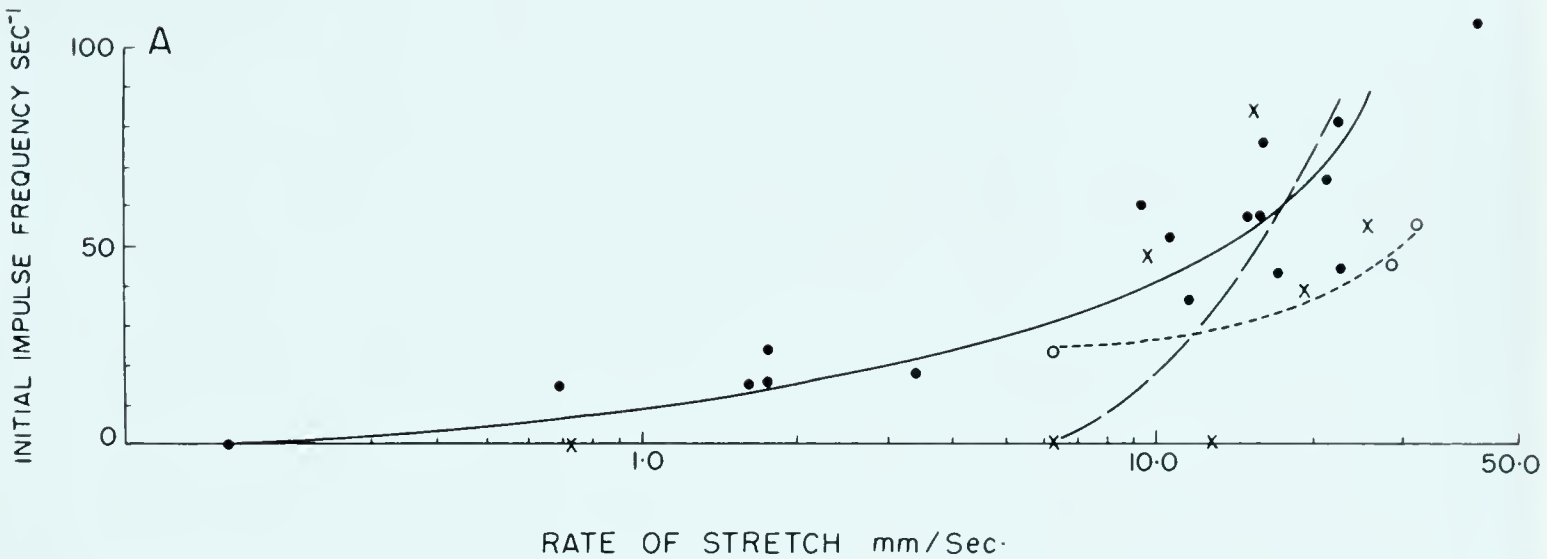


Fig. 13. A) Initial impulse frequency vs log rate of stretch for receptor 63 of experiment 59. The receptor region was stretched in three different directions:

i) solid line and filled circles - 0°
ii) broken line and crosses - 90°
iii) dashes and circles - 135°

See Table IV for directions.

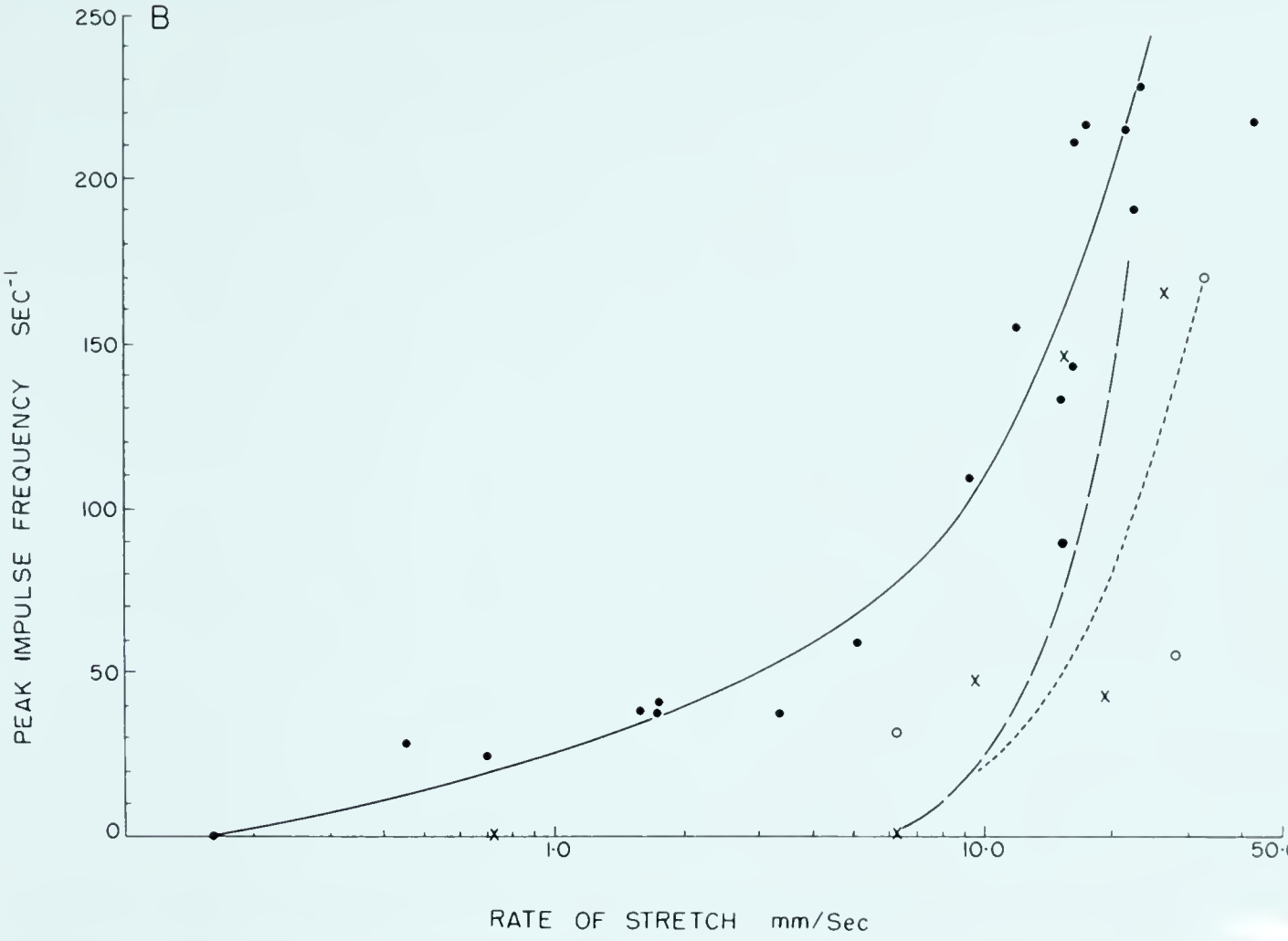


Fig. 13. B) Peak frequency vs log rate of stretch for the same receptor as in A. Notation as in Fig. 13 A.

stimulus failed to yield a discharge.

V. Comparison of Dynamic Response with a Static Response

Theory

If the system with which we are dealing is linear, it is possible to make some relatively simple predictions about it. While a rigorous discussion of a linear system is not necessary for present purposes (see Aseltine, 1958), the defining property of a linear system from which all others are derived is the principle of superposition.

Two different stimulus outputs were used in the experiments to be discussed here; 1) the ramp and 2) the step. Mathematically a step function is the first derivative of the ramp function. Under the experimental conditions used, a step stimulus is one which has a relatively short time of application compared with the decay time of the response.

Mathematically, a step stimulus can be expressed as follows:

Let $S(t)$ = step stimulus, where t = time.

Then $S(t) = 0$ when $t < 0$; (1)

and $S(t) = S$ when $t \geq 0$ (2)

where S = step amplitude, a constant.

Two ways of expressing a ramp stimulus are:

(a)

Let $R(t)$ = ramp stimulus where t = time.

then $R(t) = 0$ when $t < 0$; (3)

and $R(t) = Vt$ when $t \geq 0$; (4)

where V = ramp velocity, a constant.

(B)

Since the step stimulus, as mentioned above, is the derivative of the ramp stimulus it follows that the ramp stimulus must be proportional to the integral of the step stimulus.

This can be expressed as follows:

$$R(t) \propto \int_o^t S(t)\, dt$$

$$\text{or} \quad R(t) = k \int_o^t S(t)\, dt \tag{5}$$

where k = constant of proportionality.

However, if $S(t) = 0$ at $t < 0$, (6)

and $\int_o^t S(t)\, dt = St$ at $t \geq 0$ (7)

then it follows that

$$k = \frac{R(t)}{\int_o^t S(t)\, dt} = \frac{Vt}{St} = \frac{V}{S} \tag{8}$$

Substituting V/S for k in equation (5)

$$R(t) = \frac{V}{S} \int_o^t S(t)\, dt \tag{9}$$

By assuming that the system is linear it follows directly from equation (9) that;

$$g(t) = V/S \int_o^t f(t)\, dt \tag{10}$$

where g(t) = ramp response to a ramp of velocity V and f(t) = step response to a step of amplitude S.

A graphical example of a hypothetical case is given in Fig. 14 A and B. In Fig. 14 A is shown the response per unit of amplitude that follows a power decay of the form $f = At^{-k}$ where f = frequency

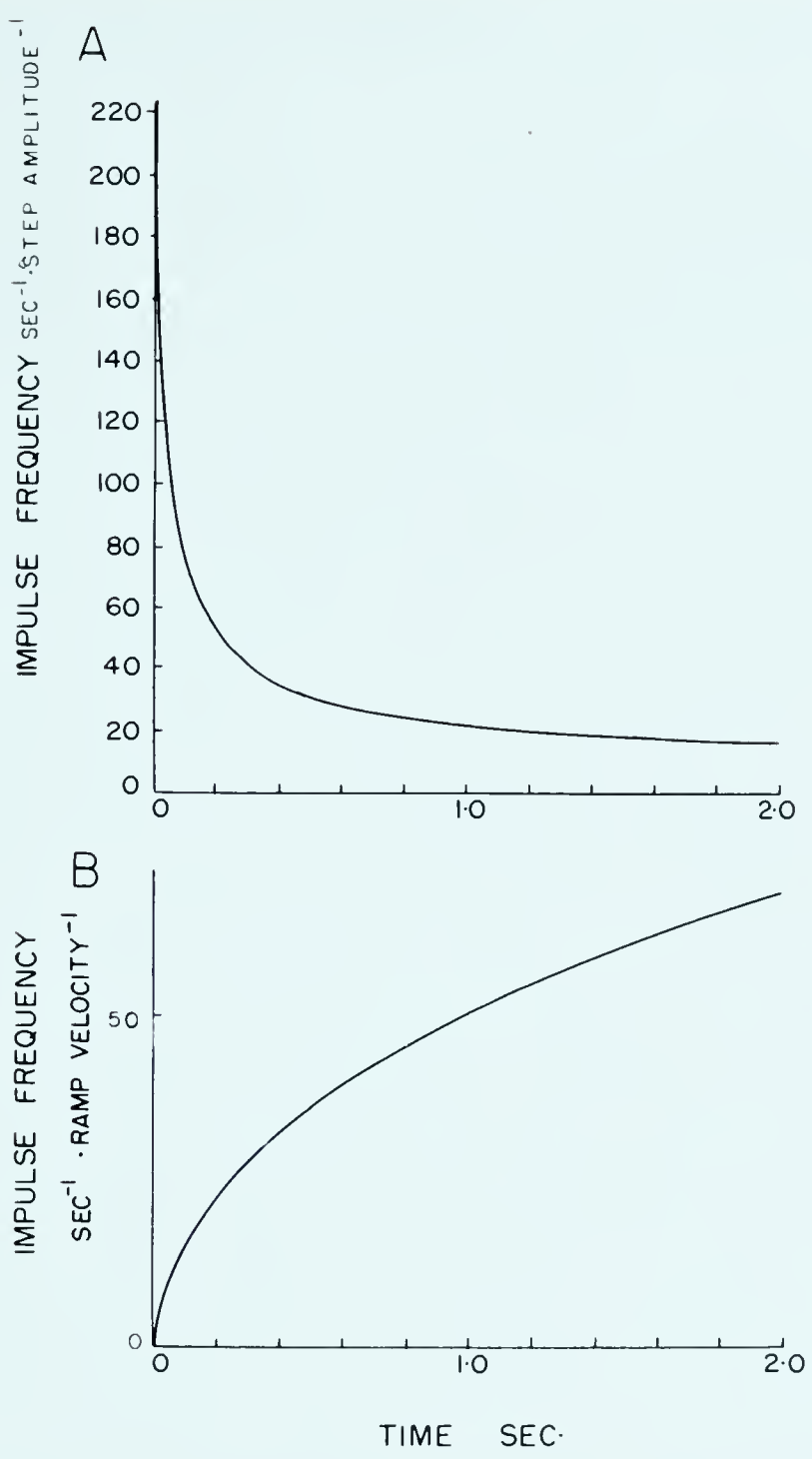


Fig. 14. A) The response of a hypothetical receptor per unit of step amplitude, whose adaptation follows a power decay of the form $f = At^{-k}$.

B) Calculated response of the same receptor per unit ramp velocity.

of discharge, A is a constant, t = time and k = a power coefficient. It follows directly from the theory that if this is the response to a step stimulus, then the response to a ramp stimulus with velocity numerically equal to the step stimulus will be given by graphically integrating the response to the step. The general equation of this curve would be;

$$g(t) = V/S \int_0^t At^{-k}\, dt$$

$$= \frac{V}{S} \quad \frac{1}{1-k} At^{1-k}$$

where V/S = unity.

This has been done using the trapezoidal rule (Granville, Smith and Longley, p. 245) and appears in Fig. 14 B.

Results

In an attempt to see if the system with which we are dealing is linear, two experiments, Nos. 51 and 59 were chosen from those performed because they had a large sampling of different stimuli for each receptor. In experiment 51 an indwelling balloon was used and in experiment 59 'tweakers' were used to stimulate the receptor.

Because of the mechanical difficulties of obtaining a true step stimulus with the experimental setup the responses of each receptor were scaled with respect to the peak frequency of each run. If the responses of a receptor when so scaled and plotted on a frequency vs time graph are superimposed on one another it shows that the receptor behaved the same way no matter how the stimulus reached a constant value. The responses of two of these receptors were scaled in this way for a number of runs and plotted on one graph for each experiment (Fig. 15 A and B). The curves for each receptor show a good number of the runs superimposed in

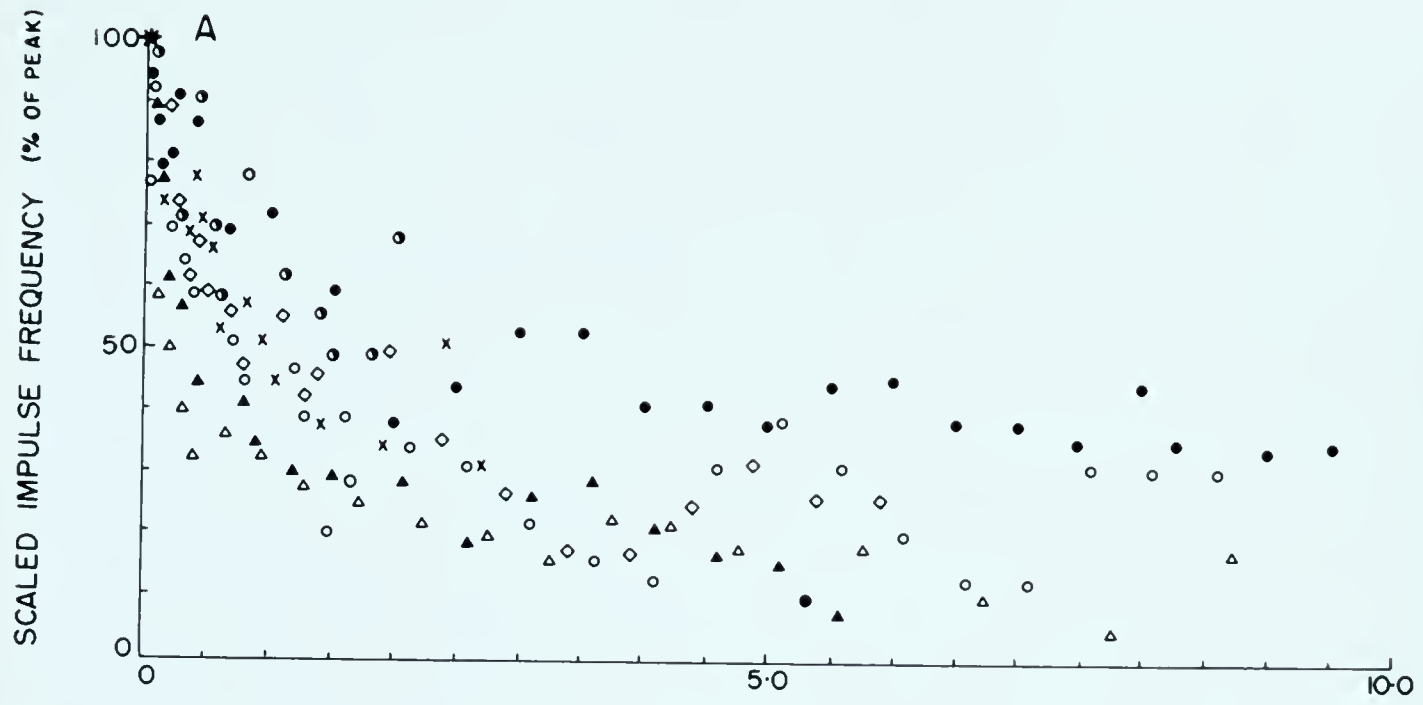


Fig. 15. Scaled adaptation plots for two receptors during periods of constant stimulus amplitude following various ramps. Time t = 0 taken as the time where the peak frequency occurred. For details see Table I.

A) Data from experiment 51, receptor 56, the ramp velocities are as follows:

○○○ run 7;
●●● run 8,
△△△ run 10,
▲▲▲ run 11,
◇◇◇ run 12,
××× run 21,
◑◑◑ run 22.

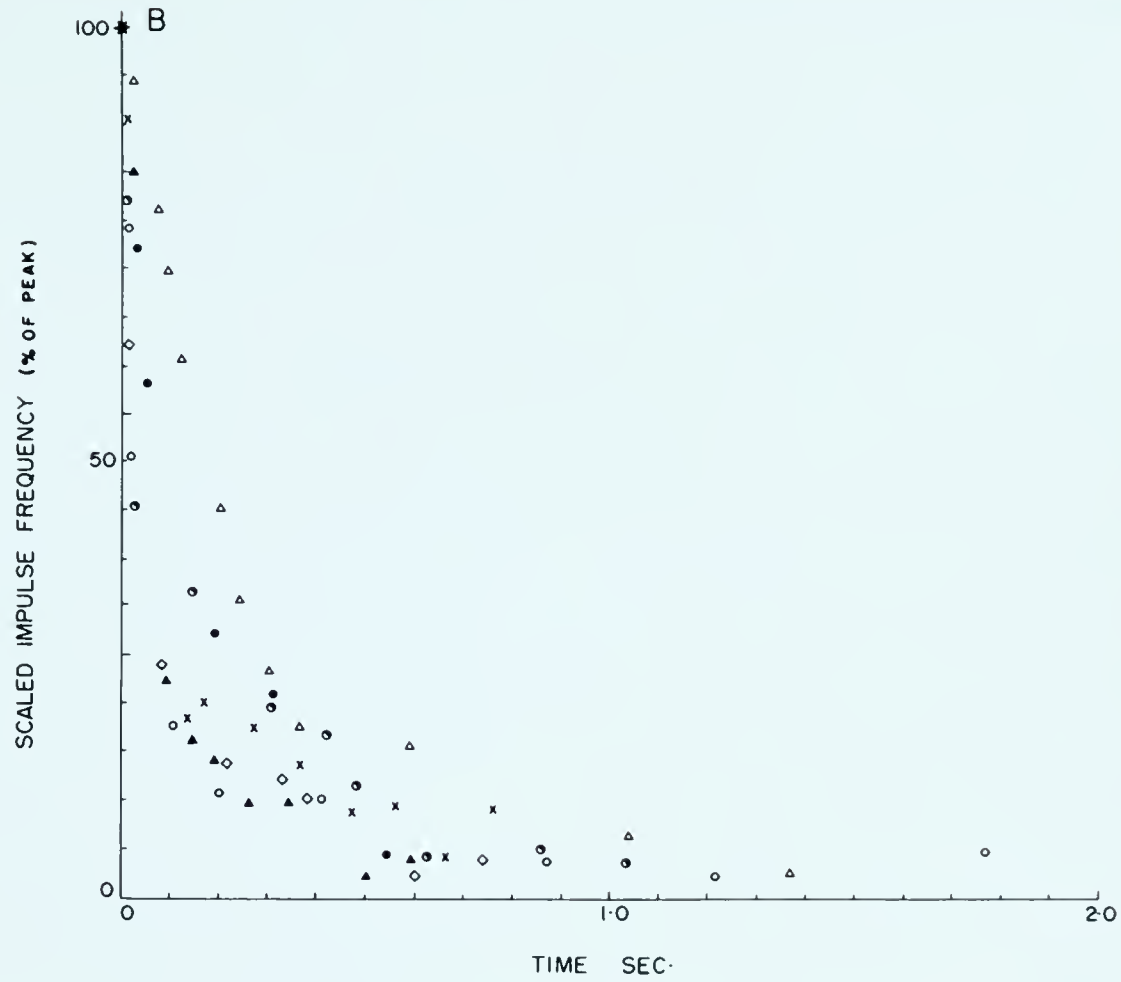


Fig. 15. B) Data from experiment 59, receptor 63. The ramp velocities are as follows;

○○○ run 1,
●●● run 2,
△△△ run 5,
▲▲▲ run 6,
◇◇◇ run 7,
××× run 11,
◑◑◑ run 27.

both the balloon and 'tweaker' experiments. It can be shown from the tables that when the response of the receptor from the peak frequency onwards is integrated and plotted as a function of time together with the scaled ramp response the two curves will not superimpose. If the system was linear then the two curves, the ramp response, and the integral of the step response should superimpose. In many of the ramp responses the frequency increased more rapidly rather than more slowly at later times during the ramp. We also tried to correlate the derivative of the ramp response with the step response or with the response after the ramp became constant, but as the statistical variation in the derivative is too large it was not constructive to do so.

DISCUSSION

Adaptation

In studying adaptation of these receptors we tried to relate the decline in frequency to a power law and to a single exponential decay but neither gave convincing results. Where possible the constants for the power law decay were calculated. The reason for choosing the power law rather than the exponential decay was mainly due to the influence of the work of Landgren (1952) and Chapman and Smith (1963). Data from the filmstrips were analysed as follows: Frequency vs time was plotted using a direct-plotting pulse frequency nomogram (Chapman, 1957), the plot values were read using an x-y plotter with time=0, chosen at the point where the stimulus began, and then plotted on log-log paper. All these measurements plus the variations in impulse frequency due to heart beat, make the determination of the constants difficult in a power law decay. Examples of how minor errors in judging both the steady state (fully adapted) impulse frequency and the exact starting time can alter the appearance of a graph of a power law decay are shown in Fig. 16. Changes of only 0.1 sec in choosing time zero from the filmstrip during some parts of the adaptation curve would result in a power law decay being changed from a straight line to a curve, making interpretation very difficult if not impossible. Similarly, changes in impulse frequency can have the same effect. These are just a few of the difficulties

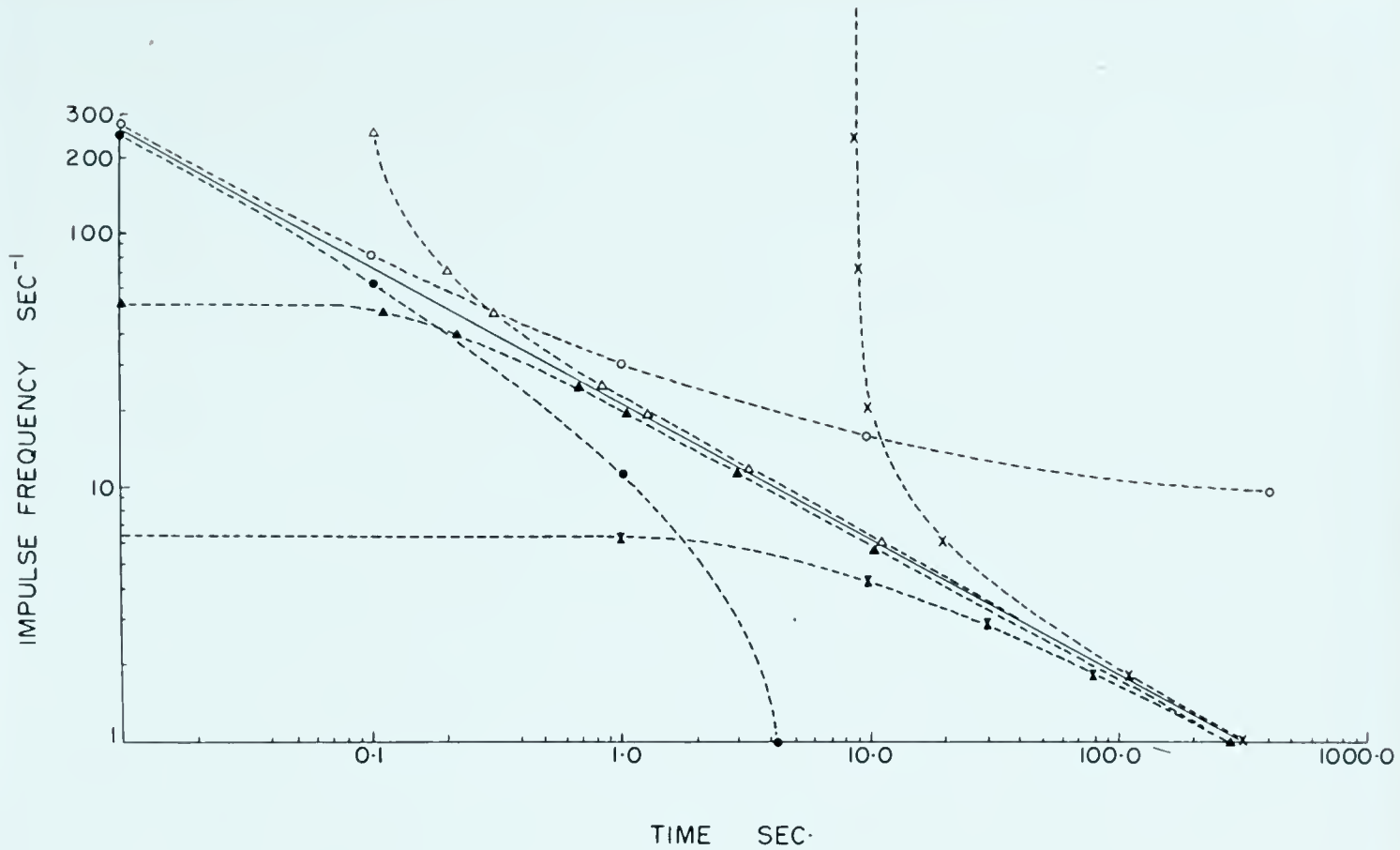


Fig. 16. The solid line represents a log-log plot of frequency *vs.* time from a hypothetical power law adaptation. The effects of shifting impulse frequencies by plus and minus 10 impulses/sec. from this curve are represented by -o- and -●- respectively; shifts in time of plus and minus 10 sec. by -x- and -⧗-, and plus and minus 0.1 sec. by -△- and -▲- .



found in analysing the data and trying to show some relationship between impulse frequency and time in the adapting receptor. Experimentally the receptors behaved in a difficult manner. In the balloon experiments as the receptor reached its adapted state the discharge became pulsatile because the heart was still beating. Therefore it was impossible to tell what it would have done in a constant stimulus situation as the stimulus was never constant. The 'tweaker' experiments presented the same problems except that the discharge became pulsatile even sooner. In no case did the discharge stay continuous throughout the cardiac cycle.

Step Amplitude and Impulse Frequency

Similar to the work of Matthews (1931) using frog muscle spindles, Landgren (1952) using cat knee joint receptors, and Loewenstein (1956) using frog skin touch receptors, we have found that cat right atrial receptors show an increased peak frequency and an increased adapted frequency to increased step amplitude. The peak frequencies attained with different receptors using similar stimuli vary considerably. Some factors which effect this inherent difference in sensitivity between receptors are the anatomical location of the receptors, differences in coupling between the receptor region and the balloon wall, and even differences in the receptor itself. This will be discussed in more detail in the section dealing with directional sensitivity.

Paintal (1953) has shown that the average impulse frequency (over a 0.5 sec. interval 1 sec. after stimulus became constant) is directly proportional to the filling volume of a vascularly isolated atrium. An attempt was made to verify this in the isolated perfused heart preparation

using an indwelling balloon to stimulate the receptor region. The responses from five separate receptors are shown in Fig. 16' in which the average impulse frequency (over the first half sec. after the end of the ramp) has been plotted against balloon volume. For comparison, Paintal's (1953) average curve from his Fig. 9 has been plotted on the same graph and appears as a heavy line in Fig. 16'. The data from our experiment 50 shows a linear relationship between balloon volume and average impulse frequency. The remainder of the experiments exhibit linearity at first, but further increasing the balloon volume causes a decrease in impulse frequency followed by a further rise. The reason we were unable in every case to show a linear rise in frequency over the entire range of balloon volumes, similar to that shown by Paintal (1953), is probably due to the mechanical coupling of the balloon to the receptor region. As the balloon reaches a certain volume it presses against the tricuspid valve and forms an extrusion through it, resulting in a reduction in the magnitude of the stimulus to the receptor region and a decrease in impulse frequency.

A somewhat similar comparison was attempted between average impulse frequency and 'tweaker' elongation but without success. According to theory, the logarithm of balloon volume is directly proportional to the logarithm of the circumference. From this it follows that a likely relationship between average impulse frequency and 'tweaker' elongation would be a logarithmic one. From the data available no such mathematical relation was evident. The most likely reason for this is the difficulty of obtaining a firm mechanical attachment between the 'tweakers' and the atrial wall.

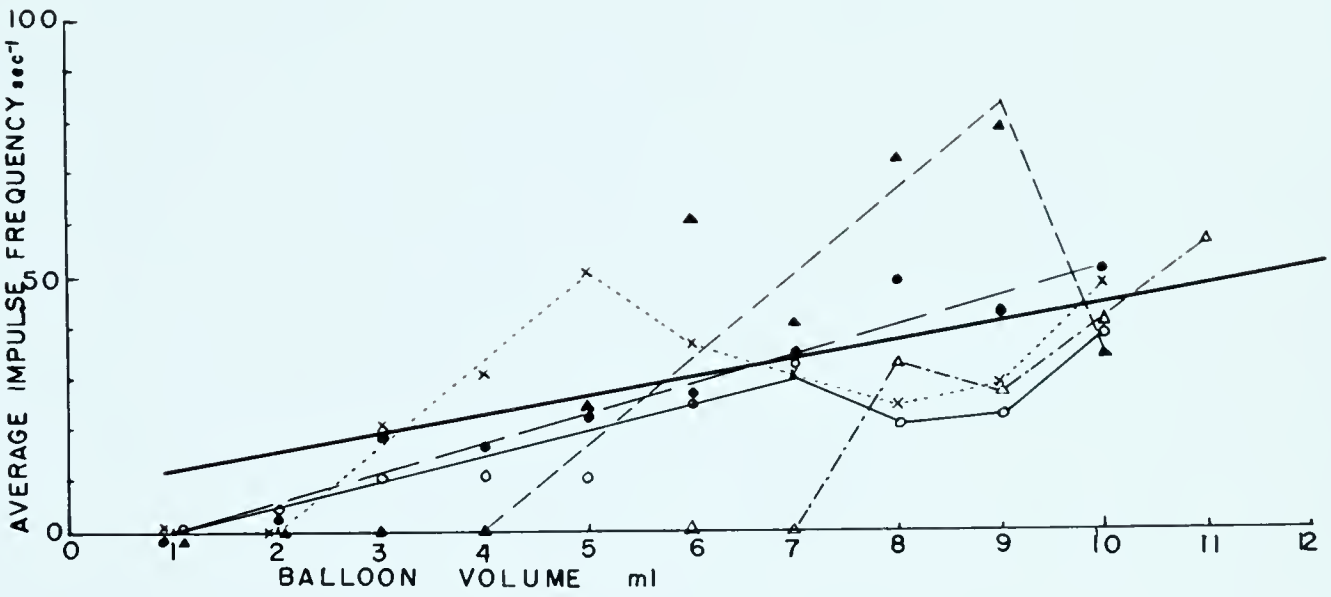


Fig. 16. Average impulse frequency over a period of 0.5 sec. commencing at the end of the ramp vs balloon volume. Data is plotted from five experiments:

×·····×	Experiment No. 34	Receptor 44
o——o	Experiment No. 48	Receptor 54
●— —●	Experiment No. 50	Receptor 55
△·—·△	Experiment No. 52	Receptor 57
▲- - -▲	Experiment No. 55	Receptor 58

The heavy line represents the average curve from Paintal's (1953) Fig. 9.

Stopped Perfusion

Stopped perfusion studies were carried out on one right atrial receptor and on one right ventricular receptor. Paintal (1953) performed stopped perfusion studies *in vivo* and reported that the atrial receptors responded

for about twenty minutes. In the two receptors studied in this section it was also found that the receptors would respond for this length of time. It was further possible to resume perfusion of the hearts and as noted in the results almost complete recovery to control levels occurred in both preparations.

It is also worth mentioning that if the receptors in the *in vitro* preparation will respond for the same length of time after stopped perfusion as those studied by Paintal (1953) *in vivo*, then it is one indication that the receptors are being maintained in relatively good condition by the perfusion technique. However, the number of experiments in both Paintal's and the present study is so small that the close agreement in survival times may merely be a coincidence.

Velocity of Stimulus

Ramps of different velocities, but with the same final magnitude of stretch, resulted in different rates of rise of frequencies, different peak frequencies, but the same frequency one second after the stimulus became constant. The greater the velocity of stimulation to the receptor, the sooner the first impulse occurred, the more rapid the increase in impulse frequency, and the higher the peak frequency. The partially-adapted frequency, as measured here, seems independent of the ramp velocity and only dependent upon the final magnitude of the stimulus.

Most of the ramps which were generated in these experiments tended to be somewhat rounded at the start and finish due to the artifacts discussed earlier. However, the results compare favourably with those of Matthews (1933), Landgren (1952), Boyd and Roberts (1953), Davis, Fowler and Lambert (1956) and Langrehr (1960), all who have studied discharge

frequencies of mechanoreceptors with ramp stimuli.

Langrehr (1960) has concluded that the mean impulse frequency is proportional to the logarithm of the velocity of stretch. While he did not explicitly define his mean impulse frequency, he seems to have taken it as one less than the number of impulses during the ramp, divided by the duration of the discharge. Five of our local stretch experiments were selected on the basis of good representation of ramp velocities. Mean frequency was plotted against ramp velocity on semi-log paper (Fig. 17) for these experiments, together with Langrehr's (1960) data which was read off his Fig. 5 (see also Fig. 13 A and B). Our results confirm those of Langrehr, although they appear to have a greater range of variability than his. However, the idea that mean impulse frequency, as defined, is directly proportional to the logarithm of ramp velocity should be treated cautiously. For example, for a given ramp velocity, the longer the ramp the more higher-frequency impulses will occur, and conversely. Langrehr (1960) does not give any data from which the percentage elongation of the receptor region can be determined. The close quantitative agreement between his ramp velocity data and ours suggests that his relative deformations must have been of the same order as ours (9 to 87%; see Table II).

Directional Sensitivity

Differences in directional sensitivity of a receptor region with local stretching were expected and were found. One obvious possibility for this is purely an experimental artifact; i.e., the receptor may have been off the axis of stretch by different amounts in each of the positions

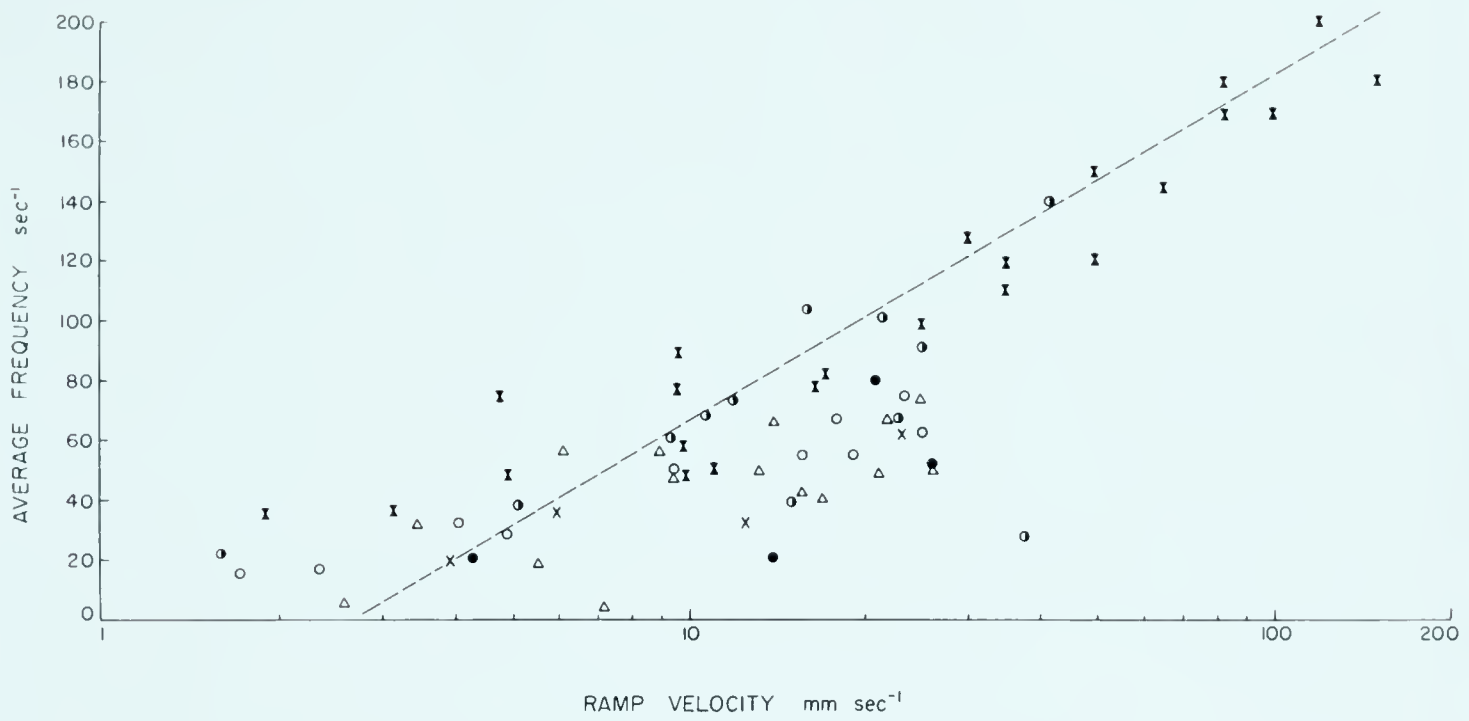


Fig. 17. Average frequency (number of impulses during a ramp less one divided by the duration of the discharge) vs ramp velocity for five experiments in which 'tweakers' were used to stretch the receptor region (o, ◑, ●, △, x) together with Lennerstrand's (1960, Fig. 5) data for cat (dashed line and XXX for his fitted curve and experimental points, respectively).

of the 'tweakers'. The tips of the 'tweakers' were about 5 mm apart at rest and the receptor region could be localized with a 1 mm probe to within about plus or minus 1 mm. Thus, considering the size of an afferent ending with its branches to be about 40 x 50 to 250 x 350 μ in diameter, different attachments of the 'tweakers' would likely result in the receptor being at varying distances from the axis of elongation. Secondly, differences in sensitivity of the receptor to differences in direction of elongation would result if equal percentage separation of the 'tweakers' tips did not result in equal deformation of the tissue between the tips of the 'tweakers'. For example, if there was some kind of folding of the tissue near the receptor endings so that in one direction, stretching would result in an unfolding of the tissue with little stretching of the receptor endings, while in another direction of stretch no unfolding, or little unfolding, may have occurred before the receptor endings were stretched. Similar differences in sensitivity would result if the tissue exhibited anisotropic elastic properties (unequal elasticity in different directions). Finally, a third reason why directional sensitivity might occur is that the receptor endings themselves might have anisotropic properties. If the multibranched endings were folded in one direction and not in another (Fig. 18 A), or if the number of endings in one direction was different than in another direction (Fig. 18 B), then when the receptor region is stretched the generator membrane would undergo different degrees of strain at terminal branches. Each of the above cases indicates that for a variety of reasons the same effective stimulus is not the same as the same absolute stimulus.

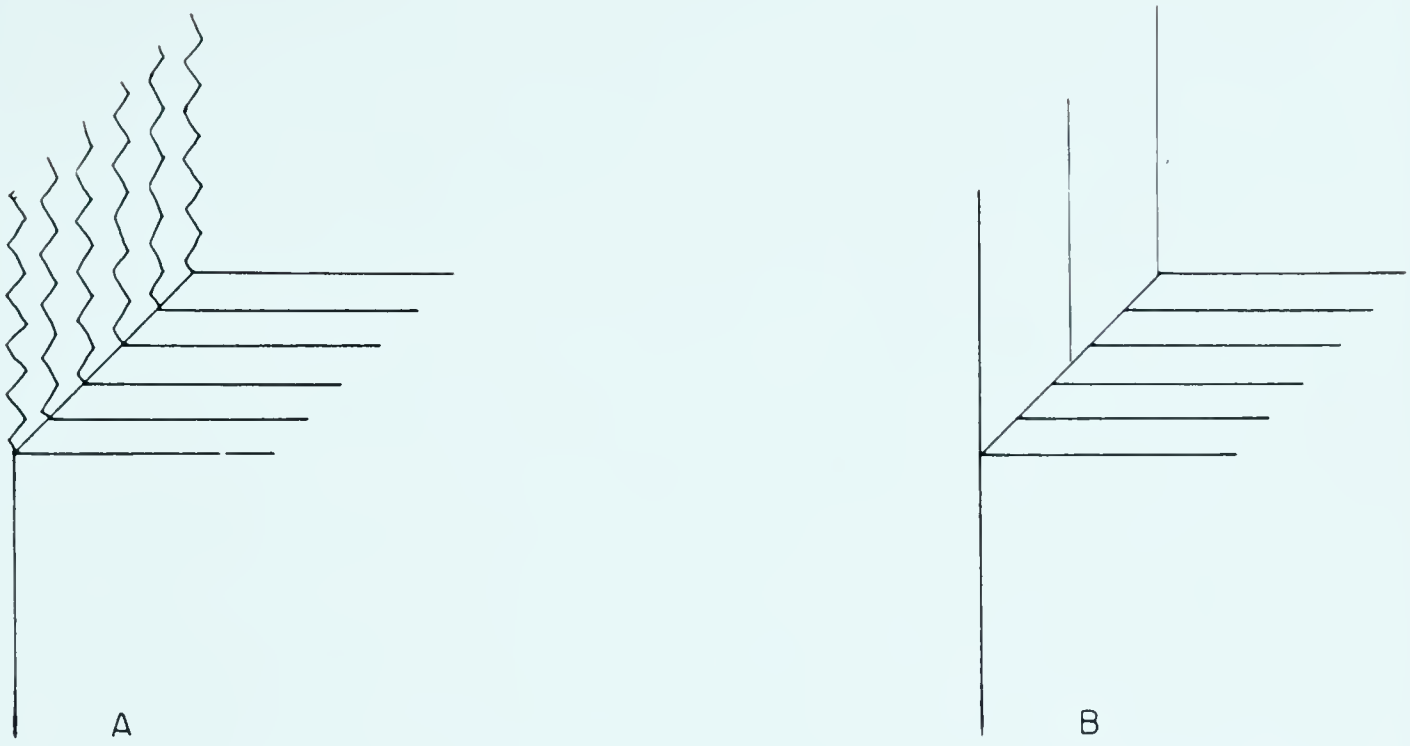


Fig. 18. Schematic diagram of hypothetical multibranched receptor endings.

A) Receptor endings folded in one direction and not in the other.

B) The number of receptor endings in one direction exceeds that in the other direction.

In addition to the findings that most receptors exhibited a directional sensitivity, the data from the experiments listed in Table IV were studied to see if there was exhibited a general directional sensitivity and/or a regional directional sensitivity common to all the receptors. In Fig. 19 peak frequency vs direction of stretch has been plotted for five receptors. In choosing the date, ramp velocity and percentage elongation of the receptor region were kept within very narrow limits so as not to effect the peak frequency. In three receptors, stretch sensitivity decreased from 0° to 150°; in another case there was no change from 0° to 90°; and in the final example, from 0° to 90° represented an increase in stretch sensitivity. Thus there is no evidence of a general directional sensitivity and/or a regional directional sensitivity. These differences in directional sensitivity are to be expected if the receptors have a complex arrangement of their terminal endings.

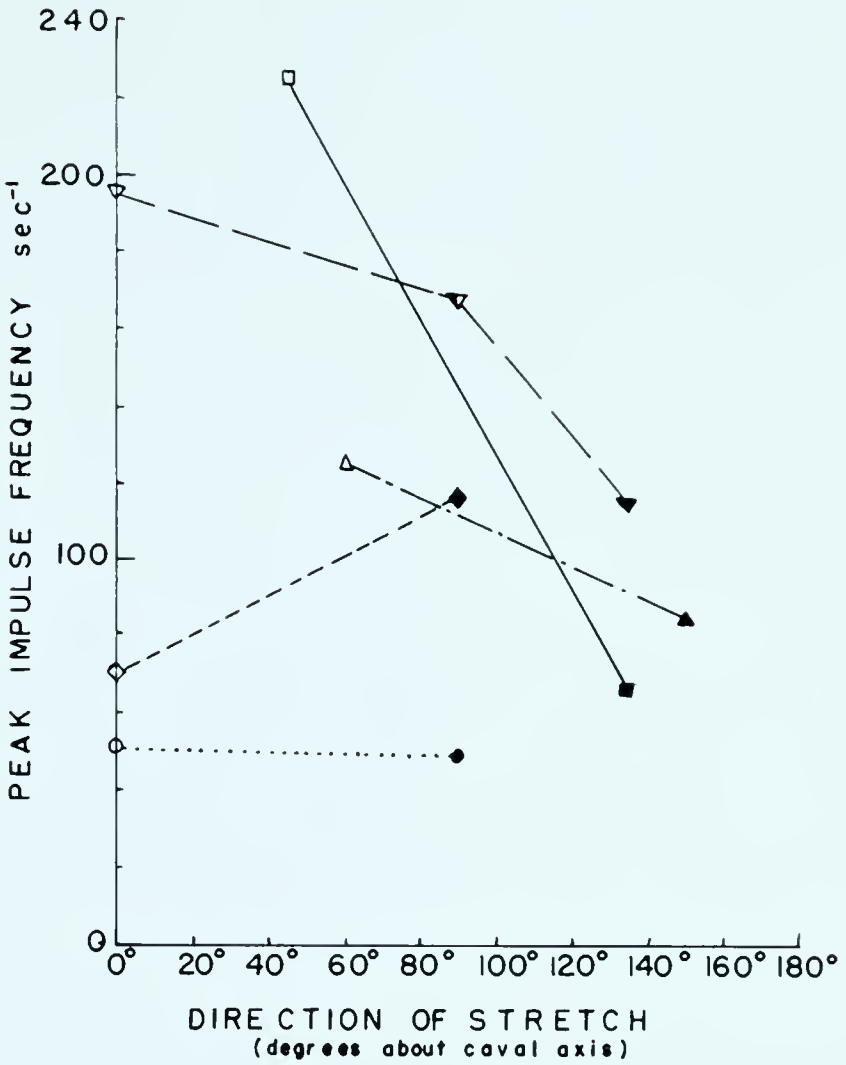


Fig. 19. Peak frequency vs direction of stretch has been plotted for the following receptors:

Receptor	Direction of Stretch (See Table IV).					
	0°	45°	60°	90°	135°	150°
59	○			●		
61		□			■	
62			△			▲
63	▽			▼	▼	
65	◇			◆		

When data from more than one run was available for a receptor the peak frequency was averaged.

Linearity Test

An attempt to show that the system being dealt with was linear was inconclusive. No attempt was made to make a rigorous mathematical presentation; however, basic principles were presented and an effort to fit the experimental data to these principles failed. If it had been possible to show that the response to a step, when integrated, yielded the response that was obtained when a ramp was applied to the same system, then we could have said that the system was linear. This did not prove to be simple; when the frequency, as a function of time, after a ramp was plotted for a receptor and compared to the integral of frequency during the ramp, no obvious relationship was evident. Incremental inflations of a balloon in three experiments with three receptors and incremental local stretching in three experiments with three receptors also failed to show that the system was linear. If it had been linear, then one simple result could have been direct proportionality between impulse frequency and step amplitude. A graph of impulse frequency as a function of step amplitude would have yielded a straight line with a definite slope with the dimensions of sensitivity. Another possible result would have been that which Matthews (1931) found, that the impulse frequency was directly proportional to the logarithm of the step amplitude. In the experiments performed none of these relationships could be verified with our results.

SUMMARY

1. The Langendorff method of perfusing isolated hearts of cats was used to study right atrial mechanoreceptors *in vitro*.
2. The location of the receptors studied has been mapped and coincides with the location reported by others.
3. The adaptation of these receptors follows the same pattern as that reported by others but no regular form of adaptation curve was obvious.
4. Increase in step amplitude results in an increased peak frequency and an increased adapted frequency.
5. The greater the velocity of the stimulus, the greater the peak frequency, the sooner the receptor responded, and the higher the initial frequency.
6. The average frequency during a ramp was found to be approximately proportional to the logarithm of the ramp velocity, rather than linear with velocity, confirming the results of Langrehr (1960).
7. Stopped perfusion studies showed a relative insensitivity to anoxia in two cardiac receptors, which responded for twenty minutes and fifty-three minutes after perfusion was stopped.
8. Differences in sensitivity of a receptor to different directions of stretch have been shown to occur at stimulating velocities up to 25 mm/sec.

9. Finally, two additional tests for linearity were attempted;

 1) comparison of the integral of the response to a step stimulus with the response to a ramp stimulus, and

 2) incremental balloon inflations and incremental elongation with 'tweakers'.

 Both also showed deviations from simple linearity.

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APPENDIX

Table I

Run Number	Balloon volume cc.	Rate of Stimulation cc/sec	Slope of log-log plot	½ time of receptor sec
Experiment No. 17	Receptor 1			
A1	1			
2	2			
3	3			0.11
4	4		- 0.6	0.22
5	5			0.18
6	5			0.29
B1	1			
2	2			
3	3			0.39
4	4			0.12
5	5			0.05
E1	1			
2	2			
3	3			0.16
4	4			0.04
5	4			0.07
6	5			0.14
F1	5		- 0.40	0.14
2	5			0.10
3	5			0.07
4	5			0.10
5	5			0.12
Experiment No. 18	Receptor 3			
D1	7			0.09
1'	7			0.11
1"	7			0.10
2	1			
3	2			
4	3			
5	4			0.06
6	8			0.19
7	0			
8	1			
9	2			
10	3			
11	4			
12	5			
13	6			0.27

Table I - continued

Run number	Balloon volume cc.	Rate of Stimulation cc/sec.	Slope of log-log plot	$\frac{1}{2}$ time of receptor sec.
Experiment No. 18	Receptor 3 - continued			
D14	0			
15	1			
16	2			
17	3			
18	4			
19	5			
20	6			0.08
21	7			0.16
22	8			0.17
E 1	4			
2	4			
3	8			0.13
4	8			0.27
Experiment No. 18	Receptor 4			
F1	9			0.26
2	1			
3	2			
4	3			
5	4			
6	5			
7	6			
10	1			
11	2			
12	3			
13	4			
14	5			
15	6			0.23
16	7			0.32
17	8			0.42
18	9			0.21
Experiment No. 18	Receptor 5			
G2	1			
3	2			
4	3			
5	4			
6	5			
7	6			
8	7			
9	8			
10	9			
11	9			0.18
12	9			0.40

Table I - continued

Run number	Balloon volume cc.	Rate of stimulation cc/sec.	Slope of log-log plot	$\frac{1}{2}$ time of receptor sec.
Experiment No. 26 Receptor 30				
A1	1			
2	2			
3	3			
4	4	0.9		
5	5	7.9		
6	6	3.0		
7	7	11.7		
8	8	9.5		
9	8	12.8		
10	9	14.9		0.32
11	10	28.6		0.63
B1	1			
2	2			
3	3			
4	4	7.6		
5	5	13.9		
6	6	17.9		
7	7	9.5		
8	8	15.7		
9	9	14.4		
10	10	22.2		
Experiment No. 26 Receptor 31				
A1	10	17.9		
2	12	16.0		0.21
3	12	19.7		0.22
4	12	20.4		0.14
Experiment No. 26 Receptor 34				
A1	10	13.0		0.59
2	1			
3	2			
4	3			
5	4	4.8		
6	5	7.3		
7	6	11.3		
8	7	7.2		0.22
9	8	11.1		0.86
10	9	15.6		0.59
11	10	14.7		0.38
12	11	18.0		0.55
13	12	24.5		0.28
14	10	13.9		0.48

Table I - continued

Run number	Balloon volume cc.	Rate of stimulation cc/sec.	Slope of log-log plot	½ time of receptor sec.
Experiment No. 26	Receptor 35			
B1	10	30.3		0.27
2	11	26.9		0.26
3	12	10.9		0.43
Experiment No. 28	Receptor 37			
A1	10	15.6		0.19
2	9	14.5		0.88
3	8	13.8		0.32
4	7	13.2		0.24
5	6	9.2		
7	5			
8	4			
9	3			
10	2			
11	1			
Experiment No. 34	Receptor 44			
A1	10	14.5	-1.2	0.12
2	1			
3	2			
4	3	5.5		0.14
5	4	5.6		
6	5	9.1		0.18
7	6	10.0		
8	7	11.9		0.13
9	8	9.0		
10	9	16.1		
11	10	25.0		
	% Elongation	mm/sec.		
B1	46	11.1		0.21
2	14	5.9		
3	35	5.6		0.24
4	42	10.3		
Experiment No. 35	Receptor 45			
B1	18	13.0		
2	33	7.2		
3	30	4.2		
4	24	8.3		

Table I - continued

Run number	% Elongation	Rate of stimulation mm/sec.	Slope of log-log plot	½ time of receptor sec.
Experiment No. 35 Receptor 45 - continued				
B5	60	10.8		
6	78	12.3		0.41
7	30	4.5		
8	75	8.9		0.57
9	87	27.4		0.21
Experiment No. 39 Receptor 48				
	Balloon volume cc.	cc/sec.		
A1	10	18.9		
2	1			
3	2			
4	3	14.6		
5	4	13.6		
6	5	12.2		
7	6	16.2		
8	7	15.2		
9	8	20.5		
10	9	15.0		0.21
11	10	22.2	- 0.92	0.04
Experiment No. 43 Receptor 51				
A1	10	19.8		0.05
2	1			
3	2	5.4		
4	3	8.8		
5	4	15.1		
6	5	27.8		
7	6	17.1		
8	7	12.6		
9	8	19.0		
10	9	16.3		0.12
11	10	20.4	- 0.81	0.03
12	10	23.2	- 0.81	0.03
Experiment No. 44 Receptor 52				
A1	10	25.7		0.07
2	2	8.7		
3	3	21.4		

Table I - continued

Run number	Balloon volume cc.	Rate of stimulation cc/sec.	Slope of log-log plot	½ time of receptor sec.
Experiment No. 44	Receptor 52 - continued			
A4	4	4.9		
5	5	5.4		
6	6	7.5		
7	7	14.6		
8	8	15.1		
9	9	11.3		
10	10	23.8		0.08
Experiment No. 48	Receptor 54			
A1	10	20.8		0.34
2	1			
3	2	7.7		
4	3	11.1		0.05
5	4	10.8		0.05
6	5	19.2		
7	5	12.5		
8	6	18.9		
9	7	17.1		0.14
10	8	11.4		0.13
11	9	15.6		0.17
12	10	29.8		0.19
13	9	10.0		
14	8	14.4		
15	7	16.7		
16	6	17.4		
17	5	10.9		
18	4	10.0		
19	3	15.0		
20	2	15.4		
21	1	11.8		
Experiment No. 50	Receptor 55			
A1	1			
2	2			
3	3	5.2		
4	4	6.7		
5	5	6.0		
6	6	6.5		
7	7	9.0		
8	8	11.4		0.18
9	9	12.9		

Table I - continued

Run number	Balloon volume cc.	Rate of stimulation cc/sec.	Slope of log-log plot	½ time of receptor sec.
Experiment No. 50 Receptor 55 - continued				
A10	10	11.2		
11	9	10.0		
12	8	11.9		
13	7	10.8		
14	6	16.7		
15	5	7.5		
16	4	6.9		
17	3	5.7		
18	2	5.0		
19	1	4.4		
B 1	8	0.7		
2	8	0.6		
3	8	0.2		
4	8	1.4		
5	8	5.7		
6	8	6.5		
7	8	9.2		
8	8	10.7		
9	8	12.1		
10	8	16.3		
11	8	19.5		
12	8	22.2		
13	8	25.8		
Experiment No. 51 Receptor 56				
A1	1	3.7		
2	2	6.3		
3	3	9.4		
4	4	9.8		
5	5	11.9		
6	6	15.4		
7	7	15.2	- 0.52	
8	8	19.0	- 0.33	0.18
9	9	15.2	- 0.57	0.26
10	10	37.0	- 0.48	0.04
11	9	21.4	- 0.62	0.16
12	8	18.2	- 0.65	0.31
13	7	24.6		0.15
14	6	16.2		
15	5	12.5		
16	4	14.8		
17	3	11.5		
18	2	8.3		

Table I - continued

Run number	Balloon volume cc.	Rate of stimulation cc/sec.	Slope of log-log plot	$\frac{1}{2}$ time of receptor sec.
Experiment No. 51 - Receptor 56 - continued				
B19	1	4.8		
20	10	32.3	- 0.51	0.06
21	10	2.1		0.35
22	10	1.0		0.25
Experiment No. 52 Receptor 57				
A1	6	12.5		
2	7	17.5		
3	8	16.0		
4	9	17.3		
5	10	30.3		0.07
6	11	27.5		0.09
7	10	25.0		0.26
8	9	17.3		0.16
9	8	11.6		
10	7	14.0		
11	6	18.5		
13	6	15.8		
14	6	19.4		
15	7	17.9		
16	8	16.7		
17	9	20.4		0.22
18	10	13.9		0.15
Experiment No. 55 Receptor 58				
A1	1	6.7		
2	2	8.0		
3	3	9.7		
4	4	12.9		
5	5	10.6		
6	6	12.5		
7	7	7.6		
8	8	11.4		
9	9	13.4		
10	10	17.9		
Experiment No. 55 Receptor 59				
	% Elongation	mm/sec.		
C1	58	6.1	- 0.60	0.72
2	72	21.7	- 0.75	0.62
3	14	2.5		
4	29	6.5		

Table I - continued

Run number	% Elongation	Rate of stimulation mm/sec.	Slope of log-log plot	$\frac{1}{2}$ time of receptor sec.
Experiment No. 55	Receptor 59 - continued			
C5	43	9.5		
6	58	4.9		
7	43	0.3		
9	43	2.1		
11	43	6.5		
12	58	1.3		
13	58	9.8		
14	58	14.2		
15	87	2.2		
16	87	19.5	- 0.82	0.20
17	87	0.9		
18	87	0.4		
19	87	15.3		
20	87	26.1	- 0.93	0.48
D1	14	35.2		
4	14	1.5		
5	29	6.8		
6	43	4.5		
7	58	7.3		0.57
8	72	18.9	- 0.55	0.74
9	72	1.0		
10	72	1.6		
11	72	6.3	- 0.71	0.71
12	72	16.7	- 0.78	0.45
Experiment No. 56	Receptor 61			
B1	33	5.9		0.24
2	9	0.9		
3	17	3.9		
4	26	6.4		
5	26	13.9		0.12
6	35	11.2	- 0.75	0.18
7	61	11.7		
9	9	0.4		
10	9	0.8		
11	9	6.3		
12	17	2.7		
13	18	20.5	- 1.03	
14	26	1.0	- 0.90	
15	21	12.4		
17	33	1.0		
18	41	13.6		0.07
19	26	13.8		

Table I - continued

Run number	% Elongation	Rate of stimulation mm/sec.	Slope of log-log plot	½ time of receptor sec.
Experiment No. 56	Receptor 61 - continued			
B20	49	23.0	- 0.69	0.09
A1	9	1.4		
3	26	2.5		
4	30	2.2		0.14
5	9	0.5		
6	17	0.4		
7	26	0.4		
8	35	0.4		
9	9	1.2		
10	15	4.3		
11	23	11.3		0.07
12	32	2.8		0.26
13	47	23.0		0.06
14	54	32.3		0.12
15	53	0.9		
17	29	7.6		
Experiment No. 58	Receptor 62			
A1	29	23.6		0.07
2	34	15.7		
3	34	17.8		
4	32	4.9		
5	29	4.0		
6	29	1.7		
7	29	2.4		
8	29	0.5		
9	29	0.3		
10	39	15.0		
11	39	23.6		0.02
12	39	15.8		0.03
13	39	9.5		0.05
15	39	0.5		
16	49	18.9		0.05
18	63	25.0		
19	58	2.0		
20	49	0.7		
21	13	4.8		
22	10	0.8		
23	24	20.3		0.20
24	24	0.1		
25	26	6.0		0.17
26	39	28.1	- 1.00	0.08

Table I - continued

Run number	% Elongation	Rate of stimulation mm/sec.	Slope of log-log plot	½ time of receptor sec.
Experiment No. 58 Receptor 62 - continued				
A27	31	19.2	- 0.96	0.06
28	27	0.2		
29	28	9.5		0.10
30	24	0.5		
31	25	35.5	- 1.00	0.31
32	33	17.5	- 1.22	0.08
33	29	0.7		
34	31	0.8		
35	27	0.5		
36	39	18.2	- 0.92	0.13
37	37	7.1	- 0.94	0.17
38	33	0.1		
39	33	0.2		
40	33	5.0		0.15
41	41	21.5	- 0.92	0.14
42	37	16.5	- 0.92	0.23
43	31	2.6		
44	45	0.9		
45	47	20.3	- 1.18	0.21
46	57	15.3	- 0.82	0.20
47	55	2.8		
48	51	0.3		
49	10	13.5		
50	22	10.2		
51	34	21.5	- 1.51	0.06
52	62	28.4	- 0.85	0.08
53	63	18.7	- 1.58	0.14
54	71	12.9	- 2.95	0.11
Experiment No. 59 Receptor 63				
A1	22	16.0	- 0.79	0.02
2	22	15.4	- 1.17	
3	22	1.6		
4	22	1.8		
5	22	5.1		0.20
6	38	17.3	- 1.58	0.02
7	47	22.0	- 1.15	0.03
8	40	10.6		0.02
10	35	0.2		
11	46	22.0		0.03
12	46	24.8	- 0.84	0.02
13	44	0.7		
14	42	0.4		

Table I - continued

Run number	% Elongation	Rate of stimulation mm/sec.	Slope of log-log plot	½ time of receptor sec.
Experiment No. 59	Receptor 63 - continued			
A15	44	9.3		
17	51	21.4	- 1.30	0.07
18	51	41.6		0.02
19	48	0.4		
21	48	1.8		
23	55	11.8	- 1.18	0.05
24	54	22.6	- 1.42	0.04
26	51	3.8		
27	53	15.1	- 1.11	0.03
29	55	19.3		
30	56	25.7		
31	54	0.7		
32	56	6.2		
33	41	15.4		0.02
35	24	9.7		
36	7	12.8		
37	22	28.0		
38	21	32.1		0.02
39	21	2.4		
40	25	6.3		
Experiment No. 60	Receptor 64			
A1	33	17.0	- 0.42	0.42
2	33	20.7	- 0.53	0.17
3	33	0.1		
4	33	0.9		
5	29	4.3		0.40
6	34	13.7		
7	33	26.2		
Experiment No. 62	Receptor 65			
A1	21	21.3		
2	21	13.2		
3	21	2.6		
4	21	5.5		
5	21	7.1		
6	13	4.4		
7	13	2.1		
8	13	1.0		
9	13	18.9		
10	26	0.2		

Table I - continued

Run number	% Elongation	Rate of stimulation mm/sec.	slope of log-log plot	½ time of receptor sec.
Experiment No. 62		Receptor 65 - continued		
A11	26	4.3		
12	26	2.8		
13	26	16.0		
14	34	26.1		0.21
15	32	0.2		
16	34	5.3		0.26
17	35	0.1		
B18	34	4.6		
19	34	17.0		0.09
20	38	22.2		0.04
21	38	9.5		0.16
22	37	0.1		
23	38	0.9		
24	37	5.9		0.14
25	40	8.9		0.30
26	40	3.5		0.39
27	41	3.4		0.55
28	40	2.3		0.27
29	41	0.1		
30	42	25.0		0.02
31	49	16.1		0.09
32	45	0.3		
33	47	2.8		0.37
34	49	14.2		0.10
35	49	17.0		0.15
36	52	23.2		0.30
37	49	3.2		
38	52	1.3		
39	49	15.6		0.28
40	30	28.6		
41	30	2.2		
42	30	1.3		
43	17	20.3		
44	17	0.3		
45	17	1.1		

Table II

Run number	Balloon volume cc.	Peak frequency impulses/sec.	Frequency 1 sec. after start of stimulus imp/sec.
Experiment No. 17	Receptor 1		
A1	1		
2	2	21	
3	3		11
4	4	64	27
5	5	63	34
6	5	75	31
B1	1		
2	2	18	
3	3	81	19
4	4	118	39
5	5	144	43
E1	1		
2	2		
3	3	81	17
4	4	119	30
5	4	117	35
6	5	95	36
F1	5	101	39
2	5	100	39
3	5	102	35
4	5	91	33
5	5	105	36
Experiment No. 18	Receptor 3		
D1	7	143	45
1'	7	128	38
1"	7	109	32
2	1		
3	2	60	
4	3	87	
5	4	155	24
6	8	131	37
7	0		
8	1		

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulses/sec.	Frequency 1 sec. after start of stimulus imp/sec.
D9	2		
10	3		
11	4	35	
12	5		28
13	6	50	37
14	0		
15	1		
16	2		
17	3		
18	4		
19	5	31	31
20	6	108	43
21	7	93	52
22	8	87	44
E1	4	24	
2	4		
3	8	116	50
4	8	109	44
Experiment No. 18	Receptor 4		
F1	9	96	
2	1		
3	2		
4	3		
5	4		
6	5		
7	6		
10	1		
11	2		
12	3		
13	4		
14	5		
15	6	54	
16	7	48	8
17	8	64	19
18	9	107	
Experiment No. 18	Receptor 5		
G2	1		
3	2		
4	3		
5	4		

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulse/sec.	Frecuency 1 sec. after start of stimulus imp/sec.
Experiment No. 18	Receptor 5 - continued		
G6	5	66	
7	6	62	
8	7	46	16
9	8	54	
10	9	75	
11	9	80	
12	9	92	
Experiment No. 26	Receptor 30		
A1	1		
2	2		
3	3		
4	4	88	
5	5	95	
6	6	93	
7	7	76	
8	8	97	
9	8	76	
10	9	68	
11	10	72	
B1	1		
2	2		
3	3		
4	4	60	
5	5	50	
6	6	66	
7	7	65	
8	8	56	27
9	9	67	39
10	10	62	38
Experiment No. 26	Receptor 31		
A1	10	43	
2	12	112	54
3	12	100	32
4	12	140	45
Experiment No. 26	Receptor 34		
A1	10	71	46
2	1		

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulse/sec.	Frequency 1 sec. after start of stimulus imp/sec.
Experiment No. 26	Receptor 34 - continued		
A3	2		
4	3		
5	4	33	23
6	5	52	
7	6	54	23
8	7	54	31
9	8	52	39
10	9	58	35
11	10	63	39
12	11	66	41
13	12	78	43
14	10	66	43
Experiment No. 26	Receptor 35		
B1	10	76	12
2	11	109	29
3	12	85	45
Experiment No. 28	Receptor 37		
A1	10	147	68
2	9	77	58
3	8	79	19
4	7	54	
5	6		
7	5		
8	4		
9	3		
10	2		
11	1		
Experiment No. 34	Receptor 44		f 1 sec after ON=K
A1	10	123	33
2	1		
3	2		
4	3	74	
5	4	48	
6	5	85	27
7	6	45	
8	7	63	4

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 34	Receptor 44 - continued		
A9	8	114	8
10	9	81	8
11	10	63	
	% Elongation		
B1	46	74	23
2	14	19	
3	35	43	6
4	42	56	23
Experiment No. 35	Receptor 45		
B1	18	37	
2	33	41	
3	30	39	
4	24	27	
5	60	60	27
6	78	61	29
7	30	40	
8	75	63	19
9	87	81	36
Experiment No. 39	Receptor 48		
	Balloon Volume cc.		
A1	10	153	35
2	1		
3	2		
4	3	44	
5	4	11	
6	5	5	
7	6	20	
8	7		
9	8	10	
10	9	28	
11	10	191	37
Experiment No. 43	Receptor 51		
A1	10	154	26
2	1		
3	2		
4	3		

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 43	Receptor 51 - continued		
A5	4		
6	5		
7	6		
8	7		
9	8		
10	9	109	
11	10	163	23
12	10	136	26
Experiment No. 44	Receptor 52		
A1	10	152	8
2	2		
3	3		
4	4		
5	5		
6	6		
7	7		
8	8	36	
9	9	65	4
10	10	147	8
Experiment No. 48	Receptor 54		
A1	10	105	8
2	1		
3	2	25	
4	3	50	
5	4	28	
6	5	31	
7	5	58	
8	6	74	
9	7	86	
10	8	86	
11	9	62	10
12	10	81	8
13	9	65	4
14	8	63	4
15	7	75	6
16	6	50	8
17	5	55	
18	4	29	

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 48	Receptor 54 - continued		
A19	3	25	
20	2	22	
21	1		
Experiment No. 50	Receptor 55		
A1	1		
2	2		
3	3	89	
4	4	43	
5	5	49	
6	6	58	
7	7	58	
8	8	97	
9	9	81	
10	10	84	
11	9	67	
12	8	50	
13	7	39	
14	6	44	
15	5	36	
16	4	27	
17	3	15	
18	2		
19	1		
B1	8	30	
2	8	31	
3	8	27	16
4	8	35	12
5	8	40	25
6	8	47	27
7	8	48	28
8	8	47	23
9	8	46	25
10	8	47	30
11	8	44	24
12	8	40	23
13	8	39	19

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 51	Receptor 56		
A1	1	52	
2	2	52	
3	3	62	
4	4	56	
5	5	57	
6	6	57	6
7	7	61	11
8	8	62	47
9	9	91	43
10	10	206	52
11	9	133	39
12	8	78	34
13	7	63	6
14	6	59	
15	5	53	
16	4	47	
17	3	50	
18	2	48	
19	1	48	
20	10	233	62
21	10	124	50
22	10	80	
Experiment No. 52	Receptor 57		
A1	6		
2	7	64	
3	8	69	
4	9	87	
5	10	120	
6	11	143	23
7	10	96	
8	9	81	
9	8	65	
10	7	30	
11	6		
13	6		
14	6		
15	7	28	
16	8	59	
17	9	64	27
18	10	70	28

Table II - continued

Run number	Balloon volume cc.	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 55	Receptor 58		
A1	1		
2	2		
3	3		
4	4		
5	5	66	
6	6	143	
7	7	126	
8	8	122	34
9	9	175	31
10	10	140	
Experiment No. 55	Receptor 59		
	% Elongation		
C1	58	34	
2	72	47	
3	14		
4	29		
5	43	35	
6	58	31	
7	43		
9	43		
11	43		
12	58	16	
13	58	54	
14	58	55	4
15	87	42	17
16	87	76	23
17	87	27	
18	87	25	
19	87	58	22
20	87	59	17
D1	14		
4	14		
5	29		
6	43		
7	58	36	
8	72	47	
9	72		
10	72	21	
11	72	35	
12	72	49	

Table II - continued

Run number	% Elongation	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 56	Receptor 61		
B1	33	47	
2	9		
3	17	20	
4	26	43	
5	26	61	
6	35	56	
7	61	66	
9	9		
10	9		
11	9		
12	17		
13	18	44	
14	26		
15	21	31	
17	33		
18	41	66	
19	26	33	
20	49	73	
A1	9		
3	26	43	
4	30	58	
5	9		
6	17		
7	26		
8	35		
9	9		
10	15	21	
11	23	76	
12	32	58	
13	47	225	26
14	54	215	
15	53		
17	29	66	
Experiment No. 58	Receptor 62		
A1	29	93	
2	34	64	
3	34	83	
4	32	31	
5	29	54	

Table II - continued

Run number	% Elongation	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 58	Receptor 62 - continued		
A6	29	28	
7	29	46	
8	29	15	
9	29	18	
10	39	93	
11	39	159	
12	39	109	
13	39	116	
15	39		
16	49	88	
18	63	80	
19	58		
20	49		
21	13	47	
22	10		
23	24	80	
24	24		
25	26	74	
26	39	129	
27	31	114	
28	27		
29	28	85	
30	24		
31	25	114	
32	33	114	
33	29		
34	31		
35	27		
36	39	107	
37	37	99	
38	33		
39	33		
40	33	67	
41	41	112	
42	37	93	
43	31	57	
44	45	26	
45	47	118	
46	57	130	
47	55	35	
48	51		
49	10		

Table II - continued

Run number	% Elongation	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 58	Receptor 62 - continued		
A50	22	39	
51	34	74	
52	62	147	
53	63	119	
54	71	107	
Experiment No. 59	Receptor 63		
A1	22	211	
2	22	89	
3	22	38	
4	22	38	
5	22	69	
6	38	227	
7	47	206	
8	40	153	
10	35		
11	45	191	
12	46	165	
13	44	24	
14	42		
15	44	78	
17	51	216	
18	51	217	
19	48		
21	48	106	
23	55	155	
24	54	228	
26	51	108	
27	53	133	
29	55	43	
30	56	167	
31	54		
32	56		
33	41	146	
35	24	48	
36	7		
37	22	56	
38	21	171	
39	21		
40	25	33	

Table II - continued

Run number	% Elongation	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 60 Receptor 64			
A1	33	140	60
2	33	124	39
3	33		
4	33		
5	29	50	
6	34	47	
7	33	50	
Experiment No. 62 Receptor 65			
A1	21	70	39
2	21	68	39
3	21	50	39
4	21	47	39
5	21	60	21
6	13		
7	13	1	
8	13	1	
9	13		
10	26	20	1
11	26	48	25
12	26	28	1
13	26	48	29
14	34	58	8
15	32	31	31
16	34	50	28
17	35	33	33
B18	34	43	12
19	34	62	16
20	38	118	16
21	38	66	10
22	37	1	
23	38	50	20
24	37	66	4
25	40	81	23
26	40	56	13
27	41	52	9
28	40	41	6
29	41	13	
30	42	107	27
31	49	116	23

Table II - continued

Run number	% Elongation	Peak frequency impulse/sec.	Frequency 1 sec. after stimulus equals a constant imp/sec.
Experiment No. 62	Peceptor 65 - continued		
B32	45	1	
33	47	44	8
34	49	93	16
35	49	85	14
36	52	54	
37	49	48	
38	52	34	39
39	49	51	12
40	30	61	1
41	30	45	
42	30	50	
43	17	29	
44	17		
45	17	31	

Table III

Run number	Velocity of stimulation	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 17 Receptor 1				
A1				
2			21	21
3			15	
4			8	64
5			43	63
6			29	75
B1				
2			18	18
3			50	81
4			51	118
5			57	144
E1				
2				
3			45	81
4			63	119
5			45	117
6			50	95
F1			56	101
2			47	100
3			50	102
4			39	91
5			36	105
Experiment No. 18 Receptor 3				
D1			64	143
1'			105	128
1"			71	109
2				
3			60	60
4			87	87
5			108	155
6			52	131
7				
8				
9				

Table III - continued

Run number	Velocity of stimulation	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 18	Receptor 3 - continued			
D10				
11			23	35
12			16	
13			29	50
14				
15				
16				
17				
18			12	
19			23	31
20			62	108
21			77	93
22			35	87
E1			16	24
2				
3			81	116
4			62	109
Experiment No. 18	Receptor 4			
F1			66	96
2				
3				
4				
5				
6				
7				
10				
11				
12				
13				
14				
15			39	54
16			38	48
17			47	64
18			54	107
Experiment No. 18	Receptor 5			
G2				
3				
4				
5				
6			55	66

Table III - continued

Run number	Velocity of stimulation cc/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 18 Receptor 5 - continued				
G7			28	62
8			35	46
9			43	54
10			47	75
11			35	80
12			64	92
Experiment No. 26 Receptor 30				
A1				
2				
3				
4	0.9	0.48	28	88
5	7.9	0.23	33	95
6	3.0	1.13	35	93
7	11.7	0.23	41	76
8	9.5	0.29	48	97
9	12.8	0.06	71	76
10	14.9	0.10	42	68
11	28.6	0.12	50	72
B1				
2				
3				
4	7.6	1.23	39	60
5	13.9	0.31	47	50
6	17.9	0.45	43	66
7	9.5	0.66	45	65
8	15.7	0.16	54	56
9	14.4	0.27	35	67
10	22.2	0.17	25	62
Experiment No. 26 Receptor 31				
A1	17.9	0.42	29	43
2	16.0	0.37	34	112
3	19.7	0.43	35	100
4	20.4	0.35	26	140
Experiment No. 26 Receptor 34				
A1	13.0	0.20	53	71
2				
3				
4				

Table III - continued

Run number	Velocity of stimulation cc/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 26	Receptor 34 - continued			
A5	4.8	0.85	2	33
6	7.3	0.71	17	52
7	11.3	0.20	40	54
8	7.2	0.29	30	54
9	11.1	0.28	38	52
10	15.6	0.26	58	58
11	14.7	0.29	39	63
12	18.0	0.13	66	66
13	24.5	0.09	78	78
14	13.9	0.14	54	66
Experiment No. 26	Receptor 35			
B1	30.3	0.14	19	76
2	26.9	0.25	36	109
3	10.9	0.53	22	85
Experiment No. 28	Receptor 37			
A1	15.6	0.28	37	147
2	14.5	0.31	29	77
3	13.8	0.40	29	79
4	13.2	0.44	31	54
5	9.2			
7				
8				
9				
10				
11				
Experiment No. 34	Receptor 44			
A1	14.5	0.17	41	123
2				
3				
4	5.5	0.47	13	74
5	5.6	0.40	12	48
6	9.1	0.43	38	85
7	10.0	0.47	26	45
8	11.9	0.39	25	63
9	9.0	0.31	41	114
10	16.1	0.26	22	81
11	25.0	0.20	45	63

Table III - continued

Run number	Velocity of stimulation mm/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 34	Receptor 44 - continued			
B1	11.1	0.24	62	74
2	5.9	0.23	19	19
3	5.6	0.47	43	43
4	10.3	0.20	37	56
Experiment No. 35	Receptor 45			
B1	13.0	0:04	29	37
2	7.2	0.06	20	41
3	4.2	0.13	18	39
4	8.3	0.11	27	27
5	10.8	0.08	35	60
6	12.3	0.05	31	61
7	4.5	0.07	27	40
8	8.9	0.01	20	63
9	27.4	0.04	56	81
Experiment No. 39	Receptor 48			
	cc/sec.			
A1	18.9	0.22	27	153
2				
3				
4	14.6	0.48	17	44
5	13.6	0.24	11	11
6	12.2	0.29	5	5
7	16.2	0.27	20	20
8	15.2	0.36		
9	20.5	0.18	8	10
10	15.0	0.23	3	28
11	22.2	0.20	12	191
Experiment No. 43	Receptor 51			
A1	19.8	0.45	74	154
2				
3	5.4			
4	8.8			
5	15.1			
6	27.8			
7	17.1			
8	12.6			
9	19.0			
10	16.3	0.47	54	109
11	20.4	0.46	72	163
12	23.2	0.31	11	136

Table III - continued

Run number	Velocity of stimulation cc/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 44	Receptor 52			
A1	25.7	0.36	90	152
2	8.7			
3	21.4			
4	4.9			
5	5.4			
6	7.5			
7	14.6			
8	15.1	0.79	33	36
9	11.3	0.86	42	65
10	23.8	0.38	143	147
Experiment No. 48	Receptor 54			
A1	20.8	0.12	39	105
2				
3	7.7	0.21	25	25
4	11.1	0.11	7	50
5	10.8	0.13	12	28
6	19.2	0.13	31	31
7	12.5	0.07	27	58
8	18.9	0.12	63	74
9	17.1	0.10	42	86
10	11.4	0.10	23	86
11	15.6	0.09	41.	62
12	29.8	0.08	42	81
13	10.0	0.11	8	65
14	14.4	0.10	39	63
15	16.7	0.10	33	75
16	17.4	0.08	39	50
17	10.9	0.11	39	55
18	10.0	0.21	29	29
19	15.0	0.11	25	25
20	15.4	0.12	22	22
21	11.8			
Experiment No. 50	Receptor 55			
A1				
2				
3	5.2	0.61	27	89
4	6.7	0.65	34	43
5	6.0	0.46	36	49
6	6.5	0.36	25	58

Table III - continued

Run number	Velocity of stimulation cc/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 50	Receptor 55 - continued			
A7	9.0	0.34	31	58
8	11.4	0.21	31	97
9	12.9	0.25	8	81
10	11.2	0.27	9	84
11	10.0	0.29	35	67
12	11.9	0.25	31	50
13	10.8	0.32	26	39
14	16.7	0.30	23	44
15	7.5	0.36	19	36
16	6.9	0.41	19	27
17	5.7	0.63	15	15
18	5.0			
19	4.4			
B1	0.7	1.07	8	30
2	0.6	1.14	8	31
3	0.2	1.32	4	27
4	1.4	0.77	8	35
5	5.7	0.46	5	40
6	6.5	0.15	9	47
7	9.2	0.21	12	43
8	10.7	0.21	12	47
9	12.1	0.22	19	46
10	16.3	0.20	19	47
11	19.5	0.13	13	44
12	22.2	0.16	21	40
13	25.8	0.17	16	39
Experiment No. 51	Receptor 56			
A1	3.7	0.60	50	52
2	6.3	0.19	12	52
3	9.4	0.17	16	62
4	9.8	0.07	56	56
5	11.9	0.08	18	57
6	15.4	0.12	16	57
7	15.2	0.08	18	61
8	19.0	0.08	19	62
9	15.2	0.09	19	91
10	37.0	0.06	19	206
11	21.4	0.24	74	133
12	18.2	0.32	29	78
13	24.6	0.08	9	63

Table III - continued

Run number	Velocity of stimulation cc/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 51	Receptor 56 - continued			
A14	16.2	0.08	2	59
15	12.5	0.10	8	53
16	14.8	0.08	9	47
17	11.5	0.12	10	50
18	8.3	0.10	43	48
19	4.8	0.61	48	48
20	32.3	0.06	16	233
21	2.1	2.47	14	124
22	1.0	4.40	44	80
Experiment No. 52	Receptor 57			
A1	12.5			
2	17.5	1.07	38	64
3	16.0	0.56	45	69
4	17.3	0.44	87	87
5	30.3	0.23	120	120
6	27.5	0.31	82	143
7	25.0	0.37	66	96
8	17.3	0.53	62	81
9	11.6	0.88	47	65
10	14.0	1.02	30	30
11	18.5			
13	15.8			
14	19.4			
15	17.9	0.93	19	23
16	16.7	0.62	35	59
17	20.4	0.42	33	64
18	13.9	0.46	18	70
Experiment No. 55	Receptor 58			
A1	6.7			
2	8.0			
3	9.7			
4	12.9			
5	10.6	0.47	35	66
6	12.5	0.22	9	143
7	7.6	0.31	73	126
8	11.4	0.29	78	122
9	13.4	0.27	101	175
10	17.9	0.18	76	140

Table III - continued

Run number	Velocity of stimulation mm/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 55 Receptor 59				
C1	6.1	0.39	25	34
2	21.7	0.11	28	47
3	2.5			
4	6.5			
5	9.5	0.29	23	35
6	4.9	0.96	31	31
7	0.3			
9	2.1			
11	6.5			
12	1.3	2.64	15	16
13	9.8	0.10	23	54
14	14.2	0.19	21	55
15	2.2	1.09	42	42
16	19.5	0.10	43	76
17	0.9	6.78	23	27
18	0.4	17.87	25	25
19	15.3	0.09	47	58
20	26.1	0.09	31	59
D1	35.2			
4	1.5			
5	6.8			
6	4.5			
7	7.3	0.20	19	36
8	18.9	0.16	32	47
9	1.0			
10	1.6	3.68	21	21
11	6.3	0.29	24	35
12	16.7	0.09	31	49
Experiment No. 56 Receptor 61				
B1	5.9	0.18	26	47
2	0.9			
3	3.9	0.32	20	20
4	6.4	0.13	29	43
5	13.9	0.08	58	61
6	11.2	0.11	40	56
7	11.7	0.16	37	66
9	0.4			
10	0.8			
11	6.3			
12	2.7			
13	20.5	0.06	44	44

Table III - continued

Run number	Velocity of stimulation mm/sec.	Time of arrival of lst impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 56	Receptor 61 - continued			
B14	1.0			
15	12.4	0.11	31	31
17	1.0			
18	13.6	0.20	61	66
19	13.8	0.15	33	33
20	23.0	0.11	62	73
A1	1.4			
3	2.5	0.35	20	43
4	2.2	0.25	24	58
5	0.5			
6	0.4			
7	0.4			
8	0.4			
9	1.2			
10	4.3	0.27	21	21
11	11.3	0.10	54	76
12	2.8	0.16	26	58
13	23.0	0.07	82	225
14	32.3	0.04	110	215
15	0.9			
17	7.6	0.11	47	66
Experiment No. 58	Receptor 62			
A1	23.6	0.07	66	93
2	15.7	0.15	54	64
3	17.8	0.08	60	83
4	4.9	0.32	17	31
5	4.0	0.25		54
6	1.7	0.81	23	28
7	2.4	0.57	16	46
8	0.5	4.53	15	15
9	0.3	5.69	22	18
10	15.0	0.09	10	93
11	23.6	0.25	81	159
12	15.8	0.15	52	109
13	9.5	0.23	27	116
15	0.5			
16	18.9	0.10	31	88
18	25.0	0.19	40	80
19	2.0			
20	0.7			

Table III - continued

Run number	Velocity of stimulation mm/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 58	Receptor 62 - continued			
A21	4.8	0.05	8	47
22	0.8			
23	20.3	0.05	68	80
24	0.1			
25	6.0		39	74
26	28.1	0.04	72	129
27	19.2	0.07	75	114
28	0.2			
29	9.5	0.07	39	85
30	0.5			
31	35.5	0.08	38	114
32	17.5	0.07	39	114
33	0.7			
34	0.8			
35	0.5			
36	18.2	0.06	71	107
37	7.1	0.06	47	99
38	0.1			
39	0.2			
40	5.0	0.11	27	67
41	21.5	0.12	30	112
42	16.5	0.11	38	93
43	2.6	1.00	28	57
44	0.9	1.35	16	26
45	20.3	0.06	29	118
46	15.3	0.03	27	130
47	2.8	1.98	16	35
48	0.3			
49	13.5			
50	10.2	0.12	39	39
51	21.5	0.09	47	74
52	28.4	0.05	63	147
53	18.7	0.26	54	119
54	12.9	0.12	54	107
Experiment No. 59	Receptor 63			
A1	16.0	0.06	76	211
2	15.4	0.09	57	89
3	1.6	0.34	14	38
4	1.8	0.54	23	38
5	5.1	0.30	69	69

Table III - continued

Run number	Velocity of stimulation m/m/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/sec.	Peak frequency imp/sec.
Experiment No. 59 Receptor 63 - continued				
A6	17.3	0.10	43	227
7	22.0	0.08	23	206
8	10.6	0.17	52	153
10	0.2			
11	22.0	0.06	92	191
12	24.8	0.14	45	165
13	0.7	4.16	14	24
14	0.4	9.98		
15	9.3	0.17	61	78
17	21.4	0.09	70	216
18	41.6	0.06	106	217
19	0.4	2.99		
21	1.8	0.37	16	106
23	11.8	0.19	36	155
24	22.6	0.07	44	228
26	3.8	0.55	97	108
27	15.1	0.17	57	133
29	19.3	0.12	39	43
30	25.7	0.08	56	167
31	0.7			
32	6.2			
33	15.4	0.06	85	146
35	9.7	0.15	48	48
36	12.8			
37	28.0		56	56
38	32.1	0.04	65	171
39	2.4			
40	6.3	0.14	24	33
Experiment No. 60 Receptor 64				
A1	17.0	0.06	45	140
2	20.7	0.07	54	124
3	0.1			
4	0.9			
5	4.3	0.54	27	50
6	13.7	0.16	41	47
7	26.2	0.05	50	50
Experiment No. 62 Receptor 65				
A1	21.3	0.08	70	70
2	13.2	0.15	68	68
3	2.6	3.00	4	50

Table III -continued

Run number	Velocity of stimulation mm/sec.	Time of arrival of 1st impulse sec.	Initial frequency imp/ sec.	Peak frequency imp/sec.
Experiment No. 62	Receptor 65 - continued			
A4	5.5	0.32	4	47
5	7.1	0.24	47	60
6	4.4			
7	2.1	0.61	1	1
8	1.0	1.19	1	1
9	18.9	0.31		
10	0.2	16.13	6	20
11	4.3	0.41	43	48
12	2.8	0.54	19	28
13	16.0	0.33	35	48
14	26.1	0.15	56	53
15	0.2	10.36	1	31
16	5.3	0.51	39	50
17	0.1	30.33	1	33
B18	4.6	0.46	43	43
19	17.0	0.16	39	62
20	22.2	0.12	54	118
21	9.5	0.34	27	66
22	0.1	15.77	1	1
23	0.9	1.65	1	50
24	5.9	0.25	33	66
25	8.9	0.63	29	81
26	3.5	0.57	31	56
27	3.4	0.42	2	52
28	2.3	0.40	1	41
29	0.1	23.44	1	13
30	25.0	0.13	58	107
31	16.1	0.16	31	116
32	0.3	3.47	1	1
33	2.8	0.53	2	44
34	14.2	0.29	45	93
35	17.0	0.12	33	85
36	23.2	0.23	42	54
37	3.2	3.70	1	48
38	1.3	0.72	1	34
39	15.6	0.28	40	51
40	28.6	0.07	61	61
41	2.2	0.76	37	45
42	1.3	1.02	36	50
43	20.3	0.10	3	29
44	0.3	3.38		
45	1.1	0.22	4	31



Table IV

Experiment number	Receptor number	Direction of stretch	Run numbers
55	59	0° & 90°	C1 - C20 & D1 - D12
56	61	135° & 45°	B1 - B20 & A1 - A17
58	62	150° & 60°	A1 - A20 & A21 - A26; B27 - B54
59	63	0° & 90° & 135°	A1 - A27 & A29 - A36 & A37 - A40
62	65	90° & 0°	A1 - A17; B18 - B34 & B35 - B45

